Cardiac
PRESSURES AND PULSES

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Cardiac PRESSURES AND PULSES

A Manual of Right and Left Heart Catheterization

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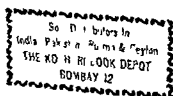
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Introduction

Catheterization of the right heart was initiated by a German surgeon (Forssmann) in 1929 It is not surprising that a surgeon, and not a physician was the first to try this technique At that time physicians were far less familiar with the use of catheters and intravenous drip methods The only surprising fact is that a surgeon rather than a urologist may claim to have been the first

While Forssmann s attempts on himself were considered at the time only an unusual technical exercise, Cournand and his group perfected this method to such a degree that it was gradually accepted and utilized by more and more researchers Dexter, Lenègre, McMichael and Burchell together with their teams further developed the techniques which are currently used

The method as originally described, was based on the oxygen determination of blood samples aspirated from the chambers of the right heart or pulmonary artery and on pressure measurements Later, catheterization of the chambers of the left heart through septal openings added interesting data to those already known Intracardiac electrocardiography determination of cardiac output by means of the Fick principle, and study of the pulmonary venous pressure by firmly wedging the catheter in a pulmonary arteriole were subsequent additions

Left heart catheterization is a relatively recent development which will very likely extend the realm of this technique to include a much larger group of patients Cases with aortic valve lesions (which are outside the realm of right heart catheterization) and cases with mitral valve lesions (in which right heart catheterization gives only indirect data) may be studied by left heart catheterization

Pressure tracings are usually taken with a conventional film speed which is inadequate for the study of accurate details of the pulse This small monograph is based on tracings recorded with a somewhat different technique aiming to study in detail various patterns of the pressure pulses of the heart and vessels

Cardiovascular physiology details of technique formulas used in catheterization, and a study of artifacts have been included in this volume to simplify the task of young cardiologists who may wish to begin working in the fascinating and ever expanding field of cardiac catheterization

The authors wish to thank Dr A B Lima, who collaborated in the study of some cases during his sojourn in Chicago in 1953–1954

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Chicago 1956

CHAPTER ONE

The Cardiac Cycle

CONTRACTION OF THE ATRIA

The cardiac cycle starts with *the contraction of the atria*, also called atrial systole[1] A wave of contraction follows that of excitation, moving downward from above,[2] thus creating a propulsive wave toward the ventricles An appreciable backflow toward the large veins is prevented by the initial contraction of the musculature at the opening of the veins and by the high venous pressure accompanying this phase

Atrial contraction takes place during that short phase which immediately precedes ventricular contraction, the *presystole* As the A V valves are open during atrial contraction, only a moderate rise in pressure takes place within the atria and the contraction is mainly revealed by movement of blood (Fig 1)

Atrial contraction is not indispensable for ventricular filling because the greatest part of this ventricular phase occurs in early diastole Still, contraction of the atria completes ventricular filling and is one of the factors upon which the normal function of the A V valves is based[3] In rapid heart action and in mitral stenosis atrial contraction may acquire a much greater importance

CONTRACTION OF THE VENTRICLES

Initiation of ventricular contraction increases the pressure in the ventricles and closes the *atrioventricular valves* (tricuspid valve in the right heart, mitral valve in the left heart) Immediately afterwards the contraction of the papillary muscles prevents an eversion of these valves and permits a further rise of pressure to a point equaling and then exceeding the pressures existing in the aorta and in the pulmonary artery In this short period the ventricular contraction builds up pressure without causing motion of blood This short phase is called *the period of tension* or *the period of isometric contraction* because the muscle fibers of the ventricles build up tension steadily even though unable to become shorter

At the onset of ventricular contraction the entire myocardium has been excited Owing to the latency between excitation and contraction however those fibers which were excited first start contracting first Then, more and more fibers contract For this reason ventricular pressure rises slowly at first then very rapidly[4]

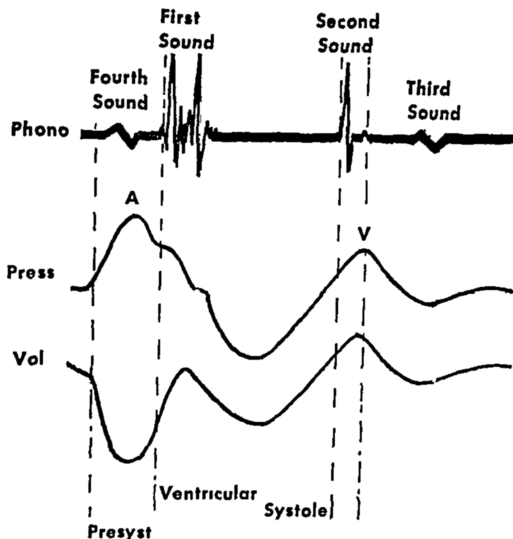


Fig 1—Changes of atrial pressure (press) and volume (vol) during the cardiac cycle and correlation with heart sounds tracing (phono)

As soon as the ventricular pressure exceeds that of the respective artery, *the semilunar valves open* and *the outflow begins* During this period of *outflow* or *ejection*, the fibrous septum which supports the A V valves is lowered by the contraction of the ventricles (papillary muscles, free ventricular wall, and intraventricular septum) *As a consequence* a remarkable increase in size of the atria takes place during systole causing suction of blood from the veins

From beginning to end the ventricular pressure maintains a steady course Still, *during the last part of ejection, outflow is more limited* Therefore, ejection has been divided into two parts *maximum ejection*, which includes about one half of the time and expels about two thirds of the blood, and *reduced ejection*, which expels about one third of the blood *in the last half of the time* [2]

MOVEMENTS OF THE VALVES

Despite their apparently delicate structure, the *flaps of the A V valves* have considerable strength and resistance When closed, they do not merely touch, but form a surface contact without folds Closure is started by the eddy currents and is increased by the ventricular contraction which immediately follows Eversion is prevented by the chordae tendineae, held by the papillary muscles The musculature of the septum and

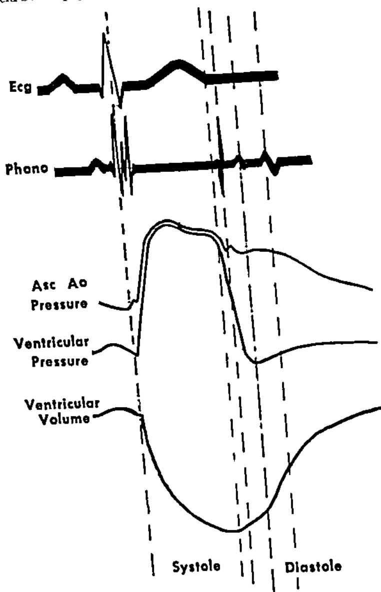


Fig 2—Changes of ventricular pressure and volume and pressure in the ascending aorta during the cardiac cycle correlation with heart sounds (phono) and electrocardiogram (ECG)

of the papillary muscles is the first to contract, insuring a timely closure of the valves The termination of atrial contraction contributes to the closure of the A V valves, because the leaflets are brought into position by the eddy currents set up by the flow through the orifices, and by a reversal of the pressure gradient This is shown by the temporary valvular insufficiency which frequently develops in cases with incomplete A V block In the event of delayed A V conduction, there may be a double closure of the A V valves the first at the end of atrial contraction, the second at the beginning of ventricular systole

Since the efflux of blood from the branches of the aorta is faster than ejection from the left ventricle, the pressure gradually declines in the aorta during the second part of systole This drop of aortic pressure may be responsible for a slight drop of pressure in the ventricular curve

The *semilunar valves of the aorta and pulmonary artery* resemble pockets attached to the wall of the vessel The blood contained in the pockets keeps the valves away from the wall Both the reversal of the gradient of pressure created by the sudden cessation of outflow and the eddy currents determine closure of these valves at the end of ventricular systole Firm attachment of the valves, muscular support from the ventricular base, and lateral apposition prevent any possibility of eversion, in spite of the lack of chordae tendineae [3]

CHANGES OF CARDIAC DIAMETERS

During ventricular contraction, *all diameters of the heart decrease* the base is pulled downward and the large vessels are stretched while the apex does not move upwards (Fig 3) The spiral arrangement of the muscular bundles of the ventricles makes their contraction very efficient, so that the blood is virtually wrung out It also causes the heart to rotate to the right, pressing the apex more firmly against the chest wall This, together

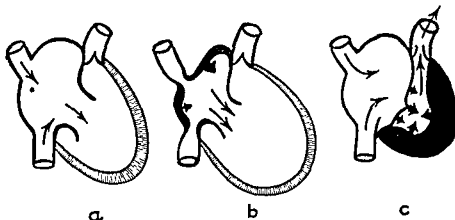


Fig 3—Schematic changes of cardiac shape during contraction (a) Mid-diastole (free flow from veins to atria and ventricles) (b) Presystole (atrial contraction completing ventricular filling) (c) Ventricular contraction (expulsion of blood into the arteries lowering of A V floor suction in the atria and veins)

with the increased firmness of the ventricular mass, is the cause of the so called *apex beat* In spite of the progressive contraction of the free ventricular walls and of the septum, a small amount of blood is left within the ventricles even in normal conditions (*residual blood*)

The *interventricular septum* seems to have an important dynamic role, a large part of right ventricular ejection seems to be due to this structure *

VENTRICULAR DIASTOLE

At the end of ventricular systole, ventricular pressure drops to zero Following an extremely brief interval of latency (so called *protodiastole*)[1] the semilunar valves of the aorta and pulmonary artery close A short time interval separates this phase from the subsequent opening of the A V valves the *isometric relaxation period* Ventricular filling starts after the end of this period e g, following the opening of the A V valves (Fig 2)

Filling of the ventricles has the following features

(a) An initial phase of *rapid passive filling* (*early diastole*) This is caused by the difference in pressure between the full atria and the empty ventricles At this time, the entire venoatrial reservoir experiences a drop in pressure due to acceleration of the stream after the opening of the atrioventricular valves

(b) A phase of *slow passive filling* (*mid diastole or diastasis*) The gradual filling of the ventricles slows down the inflow and a gradual pressure rise takes place in the venoatrial reservoir as well as in the ventricles

(c) A late phase of *rapid active filling* (*presystole*) caused by the atrial contraction which completes ventricular filling As soon as the atrial contraction is completed the ventricles start contracting because the descending stimulus has already reached the ventricular myocardium

It should be kept in mind that, during ventricular diastole, the atrium and ventricle of each side of the heart form like a single chamber

DURATION OF CARDIAC PHASES

The following time intervals in seconds may be considered typical of a normal heart with a rate of 68 beats per minute[1]

Ventricular systole		*Ventricular diastole*	
tension	0 06	protodiastole	0 04
maximum ejection	0 12	isometric relaxation	0 07
reduced ejection	0 16	rapid filling	0 10
Total	0 34	slow filling	0 22
		atrial dynamics	
		dynamic interval	0 05
		atrial contraction	0 06
		total	0 54

Because of the thinness and weakness of the *right* atrial wall and its distensibility, the filling volume of this chamber is about twice that of the *left* atrium

As atrial contraction lasts but a small fraction of the total cycle (less than one tenth of a second), the atrial wall is relaxed during most of ventricular diastole and during all of ventricular systole Thus the atria act as a reservoir for the blood coming to the heart

The traction developed by the ventricular muscles and septum on the atrioventricular junction during systole dilates the atria by causing a phenomenon of suction This is rapidly transmitted to the venous system and accelerates the flow of blood toward the atria[1] (Fig 1) This is proven not only by physiologic experiments, but also by the fact that in clinical tracings of normal subjects, recorded from the veins or atria, the pressure drops during ventricular systole

The *atrial appendages* seem to have little propulsive function and serve as complementary spaces which fill the deep niches at the base of the heart during ventricular systole

In abnormal conditions, as in cases with rapid heart rate, diastole shortens tremendously In such cases, atrial contraction may include most or all of diastole and acquire much greater importance

THE HEART SOUNDS

Auscultation of the normal heart usually reveals two sounds (or tones), occasionally, three Recording of the normal heart sounds by means of phonocardiography may reveal as many as four sounds[7]

The *first heart sound* takes place at the beginning of ventricular systole and lasts through the tension period and the beginning of the ejection period The *second heart sound* is shorter, it takes place at the end of systole, during the phases of protodiastole and isometric relaxation The name "systolic sounds" has been suggested by one of the authors for these two constantly heard sounds[8] The other two sounds, less frequently heard, take place during diastole The name "*diastolic sounds*" has been suggested for them[8]

The following dynamic phenomena take place at the time of the heart sounds

Systolic Sounds

First sound
- Initiation of ventricular systole
- Closing of the A V valves
- Opening of the semilunar valves

Second sound
- End of ventricular systole
- Closing of the semilunar valves
- Opening of the A V valves

Diastolic Sounds

Third sound Rapid passive filling of the ventricles

Fourth sound Rapid active filling of the ventricles due to atrial contraction

The mechanism of production of the **complex of the first sound** has been repeatedly investigated but the conclusions of the various researchers are by no means in agreement

Several authors suggested a purely muscular origin of the first sound Others, particularly Dock[4] and Kountz and coworkers[10] believe that the first sound is due to the sudden tension of the previously slack fibers of the A V valves A theory of mixed origin was advocated by Wiggers,[1] who denied the possibility of separating in the tracing the vibrations caused by the various structures He postulated the theory that vibrations were set up in the A V valves the chordae and the ventricular walls

The phonocardiographic studies of Orias and Braun Menendez[7] and Rappaport and Sprague[11] led to the view that the first sound is due to *four separate factors* (atrial, muscular valvular, and vascular) However, while two larger vibrations were recognized as coinciding with the two main valvular events no separate vibration or group of vibrations was found to result from muscular contraction

Experimental studies conducted by one of the authors with Alimurung and Lewis[12] proved (a) that the two main vibrations of the empty heart are extremely faint and barely appreciable and (b) that the first sound is the result of both muscular and valvular factors a sudden change in muscular tension first closing the A V valves and then opening the semilunar valves This causes a double vibration of the cardiac wall including high frequency and low frequency components which is further transmitted to the chest wall Although simultaneous with the action of the valves these vibrations are likely to arise in both the valvular and the muscular structures as a response to rapid changes in tension and pressure In other words the first sound is the audible expression of that complex movement of the heart which is also revealed by the first part of the apical thrust

The second sound complex is caused mainly by the closing of the semilunar valves and the resulting vibrations of the heart and chest wall However, vibrations of vascular origin[11] *and even the opening of the A V valves*[11, 12] contribute to its formation, at least in certain cases

The third sound arises in the ventricular wall as the result of the vibration caused by the onrush of blood at the moment of rapid passive filling of the ventricles The third sound has been attributed to valvular vibrations (a theory which is not too likely to be confirmed) or to the apical impact on the chest wall (which may be only a concurrent factor)

The fourth sound arises in the ventricular wall and is caused by the blood rushing into the ventricular chambers because of atrial contraction [7] Earlier vibrations recorded from the esophagus, can be attributed to the atrial contraction *per se*

THE ARTERIAL PULSE

The left ventricle empties itself at each beat into the aorta This vessel stores a portion of the blood received so that neither the pressure nor the flow fall too low before the next ventricular contraction The aorta offers little resistance to the flow of blood Its great distensibility, however, gives a variable resistance according to the rate at which the pressure changes, e g , the heart rate

When the aortic pressure rises suddenly during the ejection phase of ventricular systole because of sudden penetration of blood expelled from the left ventricle, the aortic volume increases considerably, creating a new space (*aortic reservoir*) When the pressure falls during ventricular diastole, the retraction of the wall can be compared to the reinjection of blood *from the reservoir into the aorta, so that the pressure tends to be maintained* in spite of the lack of flow from the heart In addition to this change in the size of the aorta, the *systolic discharge* of the left ventricle succeeds in causing a forward movement of blood The progressive expansion of the arterial wall from the center to the periphery reveals this movement [2]

The *pressure wave* caused by the contraction of the heart travels with a speed of 3–4 meters per second in the aorta and 7–14 meters per second in the peripheral arteries This speed is much greater than the average rate at which the blood flows toward the periphery (14 to 18 cm per second) [2] The difference disappears, on the other hand, in the capillaries, where the pulsating pressure is converted into steady pressure and flow

At the closure of the semilunar valves, the recoil of the aorta maintains the onward drive of the blood At this time, the peripheral arteries are still undergoing distention, but they return to a smaller size as the excess of blood flows through the capillaries

FUNCTIONS OF THE VEINS

The return of blood through the venous system is due only partly to remaining force after it has passed through one or more capillary systems Many different mechanisms have been recognized which favor the venous return (1) contractions of the veins, (2) decreasing pressure in the large veins due to the action of the heart (systolic suction), (3) aspirating effect of the low pressure existing in the thorax which increases during inspiration, and (4) action of skeletal muscles on the nearest veins

RESPIRATION

Respiratory dynamics have multiple effects on the heart and on the veins During inspiration, the diaphragm contracts and exerts pressure downwards As a result, the following changes take place

(a) The intrathoracic pressure is lowered and the intraabdominal pressure is increased favoring a flow of blood from the abdomen to the thorax

(b) The liver is compressed by the diaphragm and 'wrung out'

(c) The pericardial sac is expanded and its complementary sinuses open, favoring diastole

As a result, the blood moves from the inferior cava to the heart mainly during inspiration The blood of the superior cava, however, shows less marked changes and a more constant course Still, a remarkable inspiratory collapse of the superficial veins of the neck is frequently observed

CHAPTER TWO

Technique of Catheterization

THE TEAM

The procedure of catheterization is carried out by a special team which should include as a minimum the following persons

(a) The *chief of the laboratory of catheterization*, who prepares the vein, introduces the catheter, and manipulates it under fluoroscopic control He also supervises the work of the team

(b) A *physician* (a resident or a research assistant), who uses the recording instruments and pays special attention to the electrocardiogram of the patient and to the details of the pressure tracings throughout the entire procedure

(c) An *x ray technician* (or a research fellow), who handles the x ray apparatus according to the directions of the chief

(d) A *chemistry technician* who collects the blood samples and is responsible for the blood chemistry

If other personnel are available, another *research fellow* or *resident* may collaborate by handling the manometers and taking care of the photographic tracings

Whenever an older child (5 years or more) is catheterized, a *nurse* can collaborate by being present at the procedure, taking care of the comfort of the little patient, talking to him, and reassuring him In the case of infants or younger children, an *anesthetist* is required (the entire procedure being undertaken while the patient is under basal anesthesia) An assistant, in some cases a *surgeon*, prepares one of the branches of the femoral vein or the great saphenous vein of the right side

The catheterization team may thus include from 4 to 8 persons

The usual precautions against excessive radiation, both to the patient and the observer, are observed However, the operator wears no lead gloves and should avoid exposure of the hands to x ray

THE PATIENT

In order to obtain a basal metabolic state during the procedure, and to avoid untoward regurgitation from the stomach and other complications, a fasting condition is effected in adults and children and no feeding is given to infants for 4 to 6 hours prior to the procedure

Procain G Penicillin is given for prophylactic reasons 600 000 units are injected intramuscularly at 12 hours and at 1 hour before the procedure, the same dose is also given 24 hours after the procedure In case of hypersensitivity to Penicillin, a broad spectrum antibiotic can be given

The patient lies supine on a fluoroscopic table covered by a pad. Mild sedation is used for adolescents and adults, while infants are given basal anesthetics like *Nembutal* or *Seconal* (0.5–1.0 Gm. by rectal suppository) or *Avertin*, plus additional *Demerol* intravenously or intramuscularly, if the child becomes restless.

THE SURGICAL PROCEDURE

Right Heart Catheterization[14]

A sterile tray carries the instruments necessary for skin incision, isolation of the vein, and closure.

Any vein of either the right or the left arm can be used but the ideal site is below the confluence of the median basilic vein with the other branches running into it. Vision of the field by the patient is prevented by use of a small screen or a pillow.

The skin is sterilized and infiltrated with 1% *Novocain*, then a sterile tourniquet is applied above the elbow. A transverse incision about 1 cm. in length is made (a longitudinal cut may obviate the need for suture, but may be followed by unpleasant aftereffects). The vein is isolated by blunt dissection and a double loop of silk is passed under it. The silk is divided and one piece is pulled to the distal end of the exposed part and tied with a single knot to prevent retrograde bleeding.

While the wound is covered with sterile gauze saturated with saline the vein is elevated and a small fish mouth incision is made with a smal

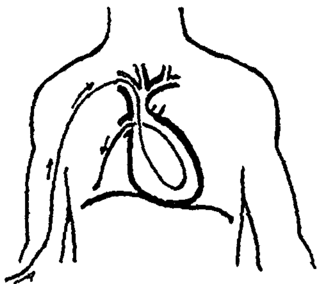


Fig. 4—The course of the catheter for right heart catheterization through a vein of the elbow.

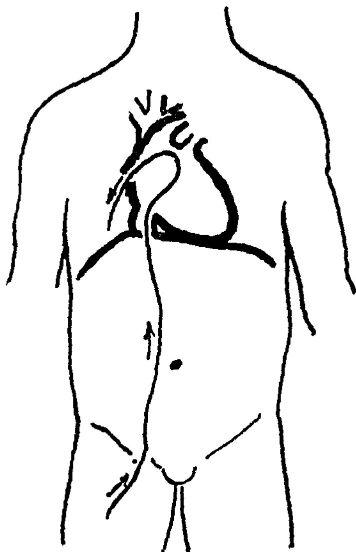


Fig 5—The course of the catheter for right heart catheterization through a vein of the lower limbs

"strabismus" scissors The catheter tip (checked for slow constant rate of dripping and lubricated with normal saline solution) is then inserted into the vein, often with the aid of a fine, curved, dressing forceps Next, it is pushed several centimeters into the lumen of the vein Further progress of the catheter is carried out under fluoroscopic control If obstruction is met on attempting to enter the chest, rotation of the catheter or slight movements of the arm and shoulder usually allow it to proceed The catheter is directed first into the superior cava, then into the right atrium (Fig 4) If the tip enters the inferior cava, the catheter should be withdrawn and reinserted

A vein of the lower limbs can also be used and is preferred whenever the veins of the arm are thin or thrombosed, or the skin is edematous or inflamed In infants, the veins at the elbow are so thin that a vein of the right inguinal region is always preferred The great saphenous vein a

branch of the femoral vein or the main femoral vein, is then prepared and used for catheterization (Fig 5) *

The catheter is passed from the femoral vein into the iliac veins, then into the inferior cava until it reaches the right atrium Once there, a complete inversion of its course is necessary for entering the opening of the tricuspid valve

A small loop is made while the catheter tip is in the right atrium We usually ask the patient to turn slightly on his left side, thus obtaining a right anterior oblique position for fluoroscopy In this position, the obscured shadow of the spine is avoided, and the exact position of the tip of the catheter (pointing forwards or backwards up or down) is accurately determined When the catheter tip is pointing anteriorly and upwards, a gentle push allows it to enter the pulmonary artery Whenever the catheter tip is facing backwards, further pushing would make a knot or a loop in the right atrium Again, if the catheter tip is pointing anteriorly, horizontally or towards the apex, pushing is likely to induce ventricular premature contractions and may create a hazard The catheter should be first slightly withdrawn then, by gentle rotation or insertion, the catheter tip can usually be passed into the pulmonary artery The tip is then pushed into one of the main pulmonary stems and further on into one of the secondary pulmonary branches (Figs 4 and 5)

As venous spasm† may prevent introduction of the catheter, it is advisable to advance the latter as far as possible and to proceed to the various pressure determinations during withdrawal It is advisable from time to time to lubricate with saline solution the surface of the catheter outside the vein especially before any further advance

The following *records* can be taken (a) pulmonary artery wedge tracing (b) main pulmonary artery tracing (c) right ventricular tracing, (d) right atrial tracing and (e) superior or inferior caval tracings

Of less common use are pressure measurements in the coronary sinus and in the renal and hepatic veins

When the catheter loop forms an acute angle within the lower part of the cardiac shadow the coronary sinus is frequently entered A blood sample taken from this point‡ and a pressure tracing taken after a slight

In order to identify the great saphenous vein a point should be found below the inguinal ligament and medial to the femoral artery according to the following description

Distance from ligament and artery

age 1–6 months 1¼ fingers (1 5–2 cm)

age 6–18 months 1½ fingers (2.5 cm)

age 1½–5 years 2 fingers (3.0–3 5 cm)

A transverse incision 1–1 5 cm long is then made across this point The great saphenous vein is usually located deeply under a longitudinal line passing through this point

† Incomplete sedation in children may be a cause of diffuse venous spasm

‡ The blood sample of the coronary sinus is much darker than that of the right atrium and has a very low oxygen content

withdrawal of the catheter will usually allow the operator to recognize this location

It is not uncommon for the catheter tip to pass through the foramen ovale or a small atrial septal defect into the left atrium, especially if the catheter is introduced from the great saphenous vein in infants Usually there is no significant evidence of left to right shunt between the two atria in such cases

In cases with atrial septal defect, the catheter can be passed from the right to the left atrium A left atrial tracing and a pulmonary vein wedge tracing may then be recorded The left ventricle may also be entered

In cases with ventricular septal defect and overriding aorta, an aortic tracing can be recorded by pushing the catheter from the right ventricle into the aorta

In cases with patent ductus arteriosus, the catheter occasionally can be passed from the pulmonary artery into the aorta

Left Heart Catheterization

Pressure tracings of the left atrium, left ventricle, and aorta have been recorded by means of different techniques based upon puncture of an *artery or of the left atrium A needle can be introduced into this chamber* through the left atrial appendage (during cardiac surgery[16]), through the main *left* bronchus,[16b, 17] from the suprasternal *fossa,*[21] or *from* the back[18–20, 22] Then a catheter can be passed into the needle and pushed following the direction of the blood stream until pressure tracings reveal that its tip has reached the desired chamber of the left heart or the aorta

Arterial puncture[23–25] This procedure is used for obtaining arterial blood, for recording arterial pressure and pulses, or for left heart catheterization

The technique for *obtaining arterial blood* requires no special instrument except a sharp No 20 gauge needle connected to a 10 cc syringe containing 0 1 cc of heparin The needle should make an angle of 45 degrees with the course of the pulsatile artery After feeling the resistance of the arterial wall, the vessel is entered without difficulty Pulsating, bright red blood then appears in the syringe and a blood sample is collected in the usual manner

When both *arterial blood* and *systemic blood pressures* are of interest, and especially if it is necessary to leave the needle in place for some time, a *Cournand arterial needle is used*

For *left heart catheterization* the technique consists of isolation and puncture of the left brachial artery with a thin walled 18 gauge needle, fitted with a stylet A very thin polyvinyl *catheter is then introduced into* the artery and gradually passed into the aortic arch *A small loop is made

* In order to visualize the transparent polyvinyl catheter one can inject 1 cc of a 70% radio opaque material into it

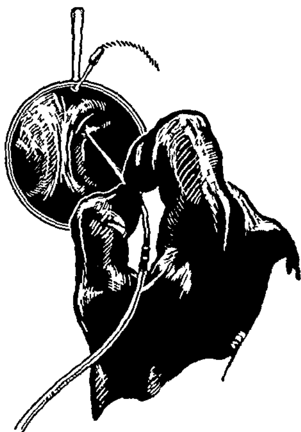


Fig 6—Transbronchial puncture of the left atrium Drawing of the view down the bronchoscope showing the carina and the needle piercing the anteromedial wall of the right main bronchus (Courtesy of Allison and Linden)

while the catheter tip rests against the aortic valve after which it can be easily pushed through the aortic valve into the left ventricle during ventricular systole The best position for entering the left ventricle is with the patient lying on his right side The procedure entails some dangers including rupture of the aorta occlusion of a coronary artery rupture of an aortic leaflet and episodes of ventricular tachycardia or flutter However, with the foregoing details of technique a gentle hand in pushing the catheter and good training with experimental animals (see below) the hazards are greatly reduced Plain catheterization of the brachial artery or the aorta should not entail any serious dangers

A *pressure tracing of the aortic arch* can be obtained not only through arterial catheterization but also through direct puncture of the arch The needle penetrates from the suprasternal notch [28]

For *left heart catheterization in animals* the animal is lightly anesthetized with intravenous nembutal A lateral position is also preferable in experimental animals because a supine position is difficult to maintain

and because it gives a better view for easy identification of the location of the catheter tip The hind legs are separated and tied to the table A large branch of one of the femoral arteries, or the artery itself, is isolated under local anesthesia A No 7 cardiac catheter is selected for larger dogs, and a No 6 for smaller dogs A bottle with a pressure of 200 mm Hg and a slow dripping method are used The catheter is introduced into the isolated artery through an incision and advanced to the arch of the aorta A small loop should be made by pushing the catheter while the tip touches the aortic valve The loop will pass as a whole through the aortic valve

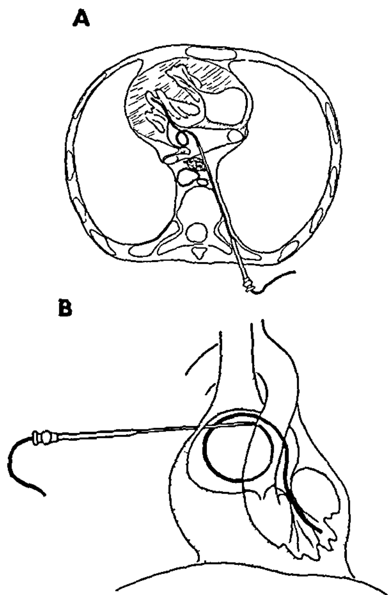


Fig 7—Left heart catheterization through the back (A) Cross sectional diagram showing a thin catheter introduced into the left ventricle through a needle placed in the left atrium (B) Sagittal plane diagram (Courtesy of Bjoerk et al Am Heart J 1954)

into the left ventricle during the succeeding ventricular systole This single maneuver decreases the possibility of damaging the aortic valve and reduces the frequency of ventricular premature beats Also, a loop is easier to make in the aorta than in the left ventricular cavity As soon as the catheter enters the left ventricle, no further attempt should be made to push it into the left atrium, unless the tip of the catheter is pointing backwards and upwards and the attempt to enter the left atrium is made *immediately after the end of* ventricular systole

Bronchial puncture[11b, 13] The patient is given mild sedation with atropine and morphine (or with demerol) Local anesthesia of the pharynx and larynx is obtained by painting the throat with *Amethocaine hydrochloride* and by instilling a few drops of this in the trachea A bronchoscope is passed down to the carina, and this is also sprayed with the local anesthetic

A needle 5 to 6 cm long having a bore of 0 3 mm is fused to a metal tube 50 cm long having a 2 mm bore This is connected with the manometer by 40 cm of polyvinyl tubing having a bore of 3 mm The needle of the cannula, connected with the manometer and filled with saline heparin solution, is introduced into the bronchoscope and, with the solution dripping, is passed through the anteromedial wall of the left main bronchus at the carina The needle penetrates about 4 cm before entering the wall of the left atrium, which is directly under the bronchus *(Fig 6)* As the procedure takes place upon a fluoroscopic table, fluoroscopic control of the position of the needle can be made before recording the tracing In the hands of several workers this procedure proved *devoid of danger*

Chest wall puncture Following x ray identification and localization of the left atrium the patient is placed on his left side on a fluoroscopic table and a skin wheal made with 1% novocaine over the ninth rib, about 5 cm to the right of the spinous process A 20 cm needle with a 1 mm bore is introduced along the upper border of the right ninth rib to the vertebral body (Fig 7A)[14, 15] The needle is then pulled back and reinserted along the vertebra until it enters the left atrium (Red blood can be aspirated at this time its color and oxygen content are compared with those of a sample obtained from the brachial artery *) Fluoroscopic examination and a typical pressure tracing confirm the correct position of the needle †

* The left atrial blood has an oxygen content which may be slightly higher than that of the brachial blood

† According to a modification of technique of Fisher[11b] the patient lies in a prone position on the fluoroscopic table The needle is inserted in one of the right intercostal spaces (from the 7th to the 9th) about 4 to 4.5 cm from the midline The needle is directed along an oblique line making an angle of 25 to 28 degrees with the sagittal plane The needle is pointing inwards and aims at reaching the heart at the center of its fluoroscopic projection This center is obtained by crossing two lines one bisects the angle of the cardiac apex and is prolonged toward the right the other bisects the angle made by the right atrium with the diaphragm and is prolonged toward the left

A thin polyethylene or polyvinyl catheter is then introduced through the needle into the left atrium It can be so manipulated that it enters first the left ventricle through the mitral valve, and then the ascending aorta through the aortic valve (Fig 7B) Typical changes of pressure reveal the passing through each valve Upon completion of the procedure, the needle should be removed first, then the catheter, in order to avoid cutting the catheter

While catheterization of the left atrium and ventricle is considered harmless, that of the aorta may cause endocardial lesions Complications caused by left atrial puncture are rare and may consist of hemopericardium, a minor asymptomatic pneumothorax, or pulmonary hemorrhage

THE CATHETERS[14]

Catheters No 5, Nos 6 and 7, short (100 cm) and long (130 cm), should be prepared * No 5 is used in infants, while one of the others is used in adults according to body size and caliber of the vein A small curvature near the tip of the catheter permits direction of its progress through rotation of the external end The latter is fitted with an adapter similar to the hub of a hypodermic needle

The catheter can be sterilized by autoclaving or by boiling for 30 minutes in distilled water After sterilization it is wrapped in a sterile towel It should be tested for surface imperfections or leaks before introduction After use, it should be rinsed with tap water, filled with "Haemosol" solution, and allowed to soak in the same solution for one hour Then it should be rinsed for one hour with tap water through a pressure connection This procedure serves to prevent deposition of particles of blood Sterilization of the catheter with antiseptics may cause febrile reactions and even pulmonary thrombosis and infarction [17]

THE INSTRUMENTS

Various apparatus are necessary for recording good pressure tracings They include

(a) A *direct writing, multi-channel cardiograph* This will be useful for recording the electrocardiogram throughout the entire procedure, for checking and calibrating the manometers, for observing the patterns of pressure, and for taking long tracings at low film speeds This apparatus may be employed *for the study of pressure levels*

(b) A *photographic, multi channel cardiograph* (a two channel apparatus may be used) This will be employed for recording sound and pressure tracings at high film speed and for the study of patterns of the pressure pulses

(c) An *oscilloscope* This will show to the operator at any given time the pattern of the pressure pulses and the electrocardiogram

* Supplied by the U S Catheter and Instrument Co of Glens Falls N Y

In our laboratory, all three apparatus are used (Sanborn Poly Viso, Twin Beam, and oscilloscope) However, a single apparatus, combining several channels a cathode ray oscilloscope, and photographic recording at various speeds, may also be used

Several different types of *manometers* can be used, including water and mercury manometers as well as the Hamilton manometer However, the need for easy recording of tracings from various chambers at different degrees of amplification, and for unlimited speed of response led to the development of special *electromanometers* and *strain gages* With these, the operator has the following advantages He can

(a) faithfully record patterns of pulses and levels of pressure from a few millimeters to several hundred millimeters of mercury

(b) determine at will the degree of amplification

(c) record either a *tracing of mean pressure* or a *pressure pulse* without distortion

(d) record the tracings with either a *direct writing* or a *photographic apparatus* and observe them on an oscilloscope

Several apparatus are available in this country including the Sanborn electromanometer and the strain gages built by Statham and by Hathaway The strain gauges are less sensitive than the electromanometers and require special amplifiers The electromanometers need a very careful—and at times painfully lengthy—balancing, before taking the records * In our experience the electromanometer is more tedious to use but more constant and more reliable than the strain gauge †

Special micro transducers to be applied at the tip of the catheter have been described by Wood[19] and by Soulié [20] The tracings shown by them are remarkably accurate However, *it is difficult* to compare the advantages or disadvantages of these *devices* with those of conventional transducers without personal experience The lack of a long column of fluid between the cardiac chamber and the transducer ought to tip the balance in favor of the *micro* transducers, if other characteristics are equal

Before starting *catheterization* the electromanometer or strain gage is prepared and calibrated The entire tubing is filled with solution 5% glucose or physiologic saline A bottle of the same solution (plus 500 units of heparin per 500 cc) is connected to a three way stopcock through a Murphy drip The bottle of the electromanometer is filled with heparinized saline solution

* The time necessary to balance the *Sanborn electromanometer* has been reduced in more recent models

† The following sentence by Burton[4] expresses an interesting viewpoint in regard to manometers For recording pressures electromanometers are most popular though some of them are annoyingly insensitive It is not sufficiently realized by physiologists (and not often admitted by radiophysicists) that the *only justification* for the use of electronics is unlimited speed of response available Where this is not a requirement physiologists are much better off with glass tubes of saline or mercury

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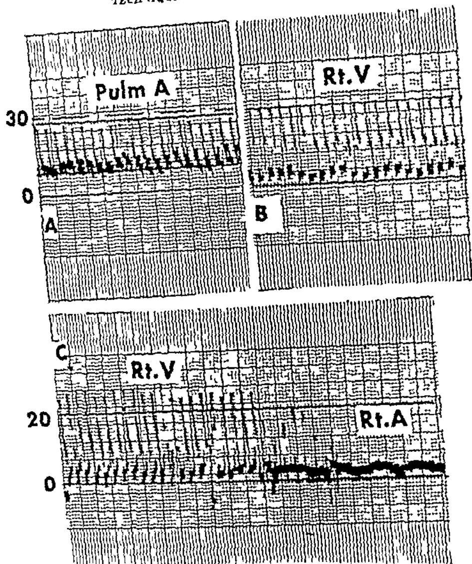


Fig 8—(A B) Pullback from pulmonary artery (Pulm A) to right ventricle (Rt V) (C) Pullback from right ventricle (Rt V) to right atrium (Rt A)

pressure pulses photographic recording and higher film speed are necessary The photographic recording insures greater fidelity and amplification in the reproduction of the details High speeds (75 mm /sec or 100 mm /sec) cause a spreading of the waves with better observation and timing of the various accidents of the tracing (Fig 11 and following) Moreover since the various simultaneous photographic tracings are not widely separated as are those of the direct writers, and since they can even cross each other timing of the waves is easy and accurate *

It should be kept in mind that the slope of any pulse wave is more or

* [illegible] and has been recently employed by Nichols et al [31]

RECORDING PRESSURE TRACINGS

The Conventional Method and Its Disadvantages

The conventional method is based on simultaneous recording of an electrocardiogram and a pressure tracing at a film speed of 25 mm /sec The amplification allowed is usually limited, resulting in waves between 5 and 15 mm high The tracing is usually taken on a direct writing apparatus

Several objections can be raised against this type of recording

(a) A wise balance between ordinate and abscissal values is necessary for accurate observation of the details of the curve and of the levels of pressure Actually, this film speed is too rapid for a quick grasp of the pressure level, it is too slow for a study of the patterns

(b) A direct writer has several disadvantages including possible distortion of the tracing (modifications due to incorrect temperature, improper balancing, improper damping, or improper pressure of the stylus) and great vertical distance between the pressure tracing and that of other cardiac tracings used for "timing" the waves

(c) The electrocardiogram, though useful for supervision of the patient, is *inadequate as a timer* on account of different "lag" between electric and mechanical phenomena,* possible lack of definition of the end of the T-wave, lack of diastolic waves (except P), and possible distorted complexes (bundle branch block, premature contractions, etc)

Therefore, our experience led us to use a somewhat different technique which is described below

Suggested Method of Recording

Film speed Two speeds should be used, according to the aim of the research Whenever one is interested in the *level of pressure* and in the *identification of the chamber* in which the catheter is placed, a low film speed should be used † Speeds of 10 mm /sec or even 5 mm /sec are usually the best They reveal clearly the typical changes in pressure when pulling back

(a) for right heart catheterization pull back from PA to RV and from RV to RA,

(b) for left heart catheterization through bronchus or chest wall pull back from AO to LV and from LV to LA

(c) for left heart catheterization through brachial artery pull back from LA to LV and from LV to AO

Direct writers are adequate for this limited purpose (Fig 8)

On the other hand, if one is interested in *the study of the patterns of the*

* A common error occurs in mitral stenosis where the first sound is delayed over the QRS complex as first proven by Cossio

† A slow film speed has been recently employed by Whitaker[53] and by Gibson and Wood[78]

Zero line Whenever one is interested in pressure data, accurate recording of the manometric zero line* is necessary before and after the tracing This requires the use of a moderate degree of amplification since, with greater amplification, the peak of the waves would go beyond the upper rim of the film (in photographic tracing) or the writing arm would be immobilized by protecting devices (with direct writers) On the other hand, if maximal amplification is required for accurate study of the patterns *one should disregard the zero line*, select the best degree of amplification, and center the beam so that both the foot and the peak of the waves are within the film† Changes of amplification should be obtained *only* through manipulation of the knobs of the electromanometer or strain gage panel (this maneuver causes no change of the baseline because of proper balancing of the instrument) and not through use of the amplifier ' knob of the electrocardiograph (this maneuver is likely to change the baseline)

It is important to employ a satisfactory standard level of zero reference with which the operator becomes acquainted in order to permit *comparison of values*

As the patients are usually in the supine position during the procedure a point which is halfway between the outermost anterior surface of the sternum and the outermost posterior surface of the chest is used It practically corresponds to the level at which both venae cavae enter the right atrium For the zero point in other positions of the patient the phlebostatic axis and the phlebostatic level described by Burch[14] are *employed*

† If one uses a photographic apparatus with a wide film (6 inches or more) both the ero line and the amplified tracing will be within the film

less steep according to the ratio of abscissa (increased by greater amplification) to ordinate (increased by greater film speed) Therefore, the best way to decide upon the rapidity of expansion of the pulse is to measure the ordinate distance between foot and peak and to relate that to the duration of systole (Fig 9) Moreover, it is important to note the distance from the peak of the wave to the second sound

Simultaneous tracings for timing We have found that the best single tracing for timing purposes is the *phonocardiogram* The exact phase of the mechanical events of the heart, and chiefly the phases of ventricular systole and diastole, can be easily identified if one uses as reference the heart sounds, simultaneously recorded by means of a phonocardiogram *Even the short phase of ventricular tension can be recognized in this way* The use of the phonocardiogram will avoid certain common mistakes, like that of attributing a slightly delayed *a wave* of an atrial pressure tracing to ventricular systole, or confusing abnormal presystolic phenomena *with those which take place during the extremely brief tension period of ventricular systole*

If more than two tracings can be recorded, the third should be either an electrocardiogram or a carotid tracing The phonocardiogram is recorded by placing a closed, radio transparent chest piece *at or slightly outside* the apex This piece is connected by a short length of hard tubing to a microphone placed under the pillow The cable of the microphone is plugged into the recording apparatus and the tracing is recorded at high speed using the "stethoscopic" method (good amplitude of the low pitched sounds)

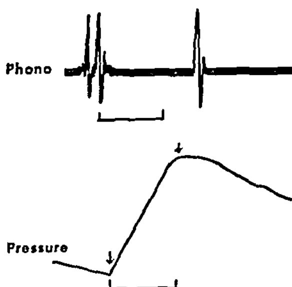


Fig 9—Measurement of the duration of the ascending slope of the pulse and correlation with duration of systole The measurement is made at the base of the pulse (distance foot to peak) and reported on the sound tracing

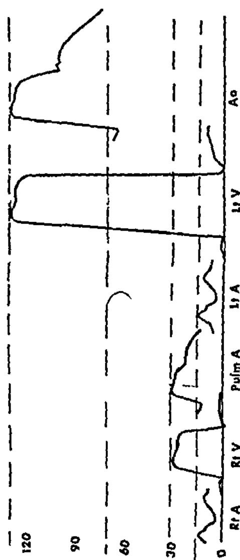


Fig 10—Normal pressures of the cardiac chambers and large arteries

CHAPTER THREE

Normal Pressures of the Cardiac Chambers and Large Vessels

Venous Pressures

A comparative study in the dog[34] revealed the following pressures, in millimeters of water

Peripheral part of right external jugular vein	39 5
Central part of same vein	−6 9
Peripheral part of thoracic superior vena cava	−9 6
Same vein near right atrium	−12 5
Inferior vena cava just below the diaphragm	5 2
Same vein below the hepatic veins	16 3
Same vein at the renal veins	31 8
Common iliac vein	40 4
Right femoral vein	52 2

According to various authors[5, 6, 31] the following pressures should be considered normal in the right heart and pulmonary artery of man (Fig 10)

Right Atrium

The pressure of the *right atrium* is higher than intrathoracic pressure but very close to atmospheric pressure In an individual lying down at rest, right atrial pressure varies between 0 and +6 mm Hg *Left atrial* pressure is similar or slightly higher However, respiration and cough affect these pressures Right atrial pressure may drop to −7 mm Hg during inspiration and rise to +8 in expiration It may rise to +60 during coughing *Mean right atrial pressure* is from 2 75[6] to 2 90[31]

Right Ventricle

The normal right ventricle has a systolic pressure of from +17 to +25 6[31] and a diastolic pressure of from 0 to +5 mm Hg Inspiration may lower the pressures to +15/−6 expiration may raise them to

On account of the interest presented by the pressures of the various waves, these have been separately calculated and the data are compared in Table II with those of Fowler et al [11] and Wynn et al

+30/+7 Cough may cause a tremendous rise, so that figures of +80 have been recorded in systole

Main Pulmonary Artery

The main pulmonary artery has a systolic pressure which should be very similar to, though slightly lower than, that of the right ventricle More marked differences may be due to technical reasons (different intrathoracic pressure at different times of recording) A slight difference in pressure, not exceeding 5 mm Hg, is to be expected between the two chambers because part of the kinetic energy of the right ventricle is absorbed by the distensible wall of the artery in systole, and returned in diastole A somewhat larger drop in systolic pressure (up to 20 mm Hg) was said to occur in cases with dilatation of the pulmonary artery * A systolic pressure between +12 and +23 should be considered normal in apnea while +10 and +29 should be the extremes of norm during respiration Diastolic pressure is markedly higher than that found in the right ventricle, due to the elastic recoil of the arterial wall, and may vary between +4 and +13 *Mean pulmonary arterial pressure* is between +10 and +18, the average being 14 4 [11]

Pulmonary Stems

The pressures are similar to those of the main artery However, here we have encountered more marked respiratory variations

Pulmonary Artery Wedge Pressure[16]

(So-called pulmonary "capillary" pressure, or pulmonary "venous" pressure, or pulmonary "capillary venous" pressures) According to most authors, this pressure should be between 7 and 13 mm Hg and should present only small variations in coincidence with the cardiac cycle

It is likely that the *pulmonary artery wedge pressure actually represents the critical closing pressure of the pulmonary arterioles* (Burton[46]) and that, whenever large systolic waves are seen, the pressure readings should be considered unreliable (too high) on account of leakage around the tip of the catheter

We have recorded cardiac and pulmonic pressures in 7 normal individuals between the ages of 5 and 48 years The data are reported in Table I Our readings show the various levels of pressure which can be found at different moments of the cardiac cycle and with different respiratory phases

* This was not true in cases with dilated pulmonary artery which were observed in our laboratory Artifacts may explain the apparent difference in pressure (see page 8?)

CHAPTER FOUR

Normal Patterns of Pressure in The Cardiac Chambers and Large Vessels

The authors have studied the patterns of pressure in 6 normal, anesthetized dogs and in 7 normal individuals Even though our description takes into consideration previous studies, emphasis is necessarily placed on our own data, because of greater accuracy of measurement attained by the different technique employed by the authors (see Chapter 2, page 20)

Considering that the patterns of the two *venae cavae* largely reflect the waves of the right atrium (with some degree of distortion), the description should start from the latter

RIGHT ATRIUM

The right atrial pattern is basically formed by two positive waves, one in presystole preceding ventricular systole, and the other during early ventricular diastole (Fig 11)

Presystolic Wave

The presystolic wave, to be called *a wave*, is caused by the *contraction* of the right atrium It starts about 0 14" to 0 15" before the beginning of the first sound in adolescents and adults, and between 0 12" and 0 14 in children Its peak always precedes the onset of the first sound, the latter usually starting during the last third or fourth of the descending branch of *a* In spite of the precedence of electrical over mechanical phenomena the peak of *a* always precedes the R wave (peak of the ventricular complex) of the electrocardiogram *In the dog* a similar relationship can be observed The onset of *a* is from 0 08" to 0 10" before the first sound and the latter starts at about the middle of the descending branch of *a* (Fig 14)

The relationship between the *a wave* and the systolic rise of ventricular pressure is well illustrated by Fig 14A If the *phonocardiogram* of the subject has a *fourth* (atrial) *sound*, this is simultaneous with the peak of the *a wave*

The *electrokymogram* of the right atrium shows a small negative wave (decrease of volume) which takes place during the positive *a wave* of the pressure tracing (increase of pressure) Both are an expression of the atrial contraction

TABLE I *Cardiovascular Pressures in Normal Humans* (Luisada and Liu)

No	Age	Sup Cava			Inf Cava			Right Atrium				Right Ventricle			Main P.A.			P A. Stem		P A Wedge
		a	S C	v	a	S C	v	a	S C	v	Mean	Syst	Diastolic Beg	Diastolic End	Syst.	Diast.	Mean	Syst.	Dia t.	
1	5	—	—	—	—	—	—	5-7	3-4	4 5	4	25	0-3	6-8	15-26	5-12	16	—	—	—
2	20	—	—	—	—	—	—	7	4	8	3 5	14-18	0-3	3-5	14-15	8-10	11	10-16	5-10	—
3	14	6	1-2	8	—	—	—	5	0-1	4	3 5	20-25	−1 + 1	2-4	20-25	6-10	13	—	—	8
4	5	—	—	—	—	—	—	—	—	—	—	28-30	0-2	3-5	24-26	8-11	17	24-26	8-11	11
5	48	7	1	4	7	1	4	7	0	3	6	22-25	0-2	5-7	—	—	—	—	—	—
6	12	—	—	—	5	1	4	4 5	−2	2 5	2 5	28	0	3	24	10	16	—	—	12 5
7	23	—	—	—	—	—	—	4 5	2 5	4 5	3 5	20	2	4	20	10	14	—	—	8

S C = Systolic Collapse

TABLE II *Average Cardiovascular Pressures in Man*

	Right Atrium				Right Ventricle			Pulmonary Artery			P A. Wedge Pressure	Left Atrium	
	a	S C.	v	Mean	Syst.	Diastolic Early	Diastolic End	Syst	Diast	Mean		a	v
Fowler et al	2 5-7	0-5	2-7 5	1-4 5	19-31 5	—	2-6	16-29	5-13	10-18	4 5-13	—	—
Luisada and Liu	5 7	1 4	4 6	3 8	23 3	+1	4 4	21 1	9 2	14 5	10	—	—
Wynn et al	—	—	—	—	—	—	—	—	—	—	—	2-8	1-9

S C = Systolic Collapse

Systolic Events

At the beginning of ventricular systole, a small notch in the pressure tracing reveals the closure of the tricuspid valve Instead of a notch, there may be only a slower *drop* of the pressure curve, or even a second (small) peak *during* the first sound We suggest that this notch or peak be represented by the symbol *av* (atrioventricular) since the letter *c*, frequently employed by others, was originally used to describe the *c wave* of the venous tracing and *c* stands for *carotid* according to the description of Mackenzie In regard to the electrocardiogram, *av* falls during the segment RS of the ventricular complex, or at the peak of S

Another small, positive wave may occur later, *after* the end of the first sound (or during the end of that sound, if it is abnormally prolonged) This systolic notch is simultaneous with the rise in pressure of the aorta and is probably due to shaking of the right atrial wall by the ascending aorta This notch, which can be called *c*, is far from being constant

Most of the ejection phase is accompanied by a *decrease in pressure* (Figs 1, 11) which is due to lowering of the A V floor and the tricuspid valve (page 6 and Fig 3) as a result of the powerful pull of the ventricular septum and the right ventricular wall It may be called *systolic collapse*

Diastolic Wave

The ascending branch of the systolic collapse rises gently during the last part of ejection on account of abundant venous inflow into the atrium A new peak is reached from 0 04" to 0 07" *after* the main vibration of the second sound at the time of the *opening* of the tricuspid valve (Fig 11) It is apparent that this opening causes a sudden change in pressure in the atrium by ' removing the bottom' of the chamber This peak may be called the *v wave* The peak of *v* is usually lower than that of *a* but may be equal to it and is never higher in normal individuals The *v wave* peak *follows* the end of the T wave of the ECG

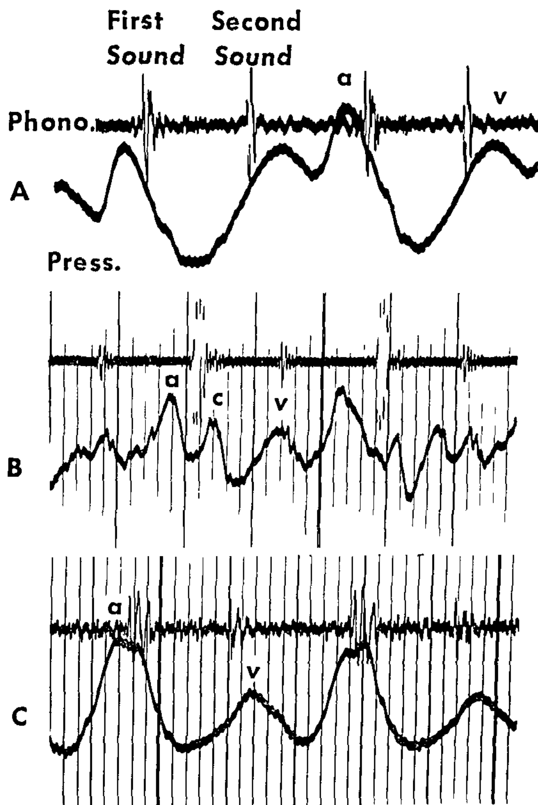


Fig 11—Patterns of pressure of the right atrium in normal subjects
(A) 5 yr old child
(B) 14 yr old girl
(C) 22 yr old girl
a = presystolic wave *av = early systolic wave v = early-diastolic wave*

SUPERIOR VENA CAVA

The pressure tracing of the superior cava is similar to that of the right atrium (Fig 12) Superimposed tracings show certain differences between RA and cavae (Fig 13)

(a) A slight delay of the a and v waves due to the time necessary for their transmission from the atrium to the cava

(b) A prominent c wave which is probably due to a systolic impact of the ascending aorta on the cava

(c) The possible lack of a separate notch for the a_t wave

INFERIOR VENA CAVA

The pressure tracing of the inferior cava is quite similar to that of the superior cava However, the peak of a is frequently more delayed than in the latter so that it may fall at the beginning of the first sound There may be absolutely no evidence of the notches a_t and c at the beginning of systole (Fig 12) The delay between right atrial and inferior caval waves is greater than for the superior cava (Fig 13)

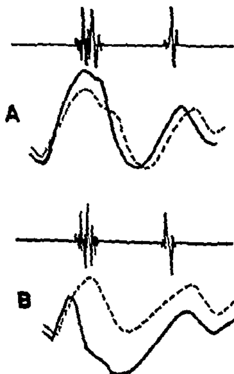


Fig 13– Relationship of caval to atrial pressure in normal subject (*from tracings of Figs 11 and 12*)

(A) Continuous line = RA Stippled line = SVC
(B) Continuous line = RA Stippled line = IVC

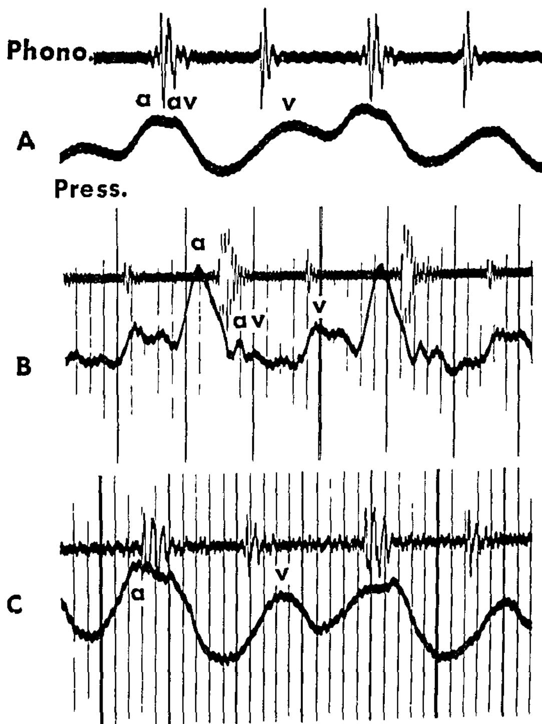


Fig 12—Patterns of pressure of the venae cavae in normal subjects
(A) 5 yr old child—inferior cava
(B) 14 yr old girl—superior cava
(C) 22 yr old girl—superior cava
a = presystolic wave v = early-diastolic wave

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The pressure tracing of the superior cava is similar to that of the right atrium (Fig 12) Superimposed tracings show certain differences between RA and cavae (Fig 13)

(a) A slight delay of the *a* and *v* waves due to the time necessary for their transmission from the atrium to the cava

(b) A prominent *c* wave which is probably due to a systolic impact of the ascending aorta on the cava

(c) The possible lack of a separate notch for the *av* wave

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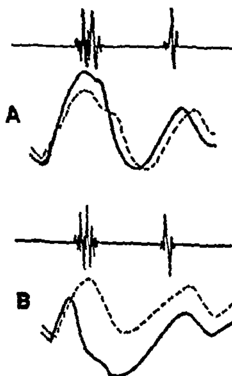


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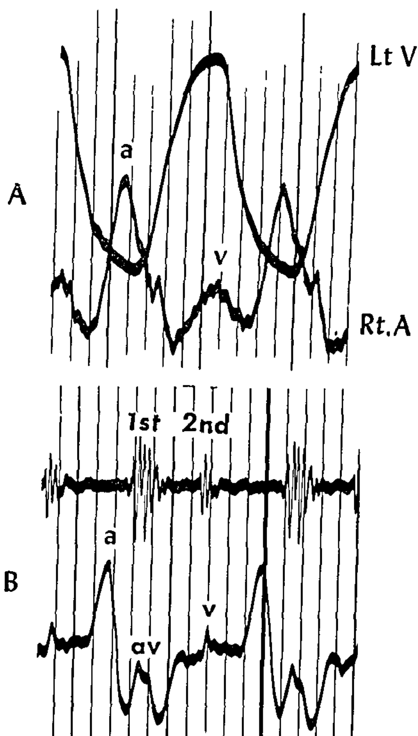


Fig 14—Right atrial tracings in two normal dogs

(A) Upper line—Left ventricular pattern Lower line—Right atrial pattern

(B) Phonocardiogram and right atrial pulses

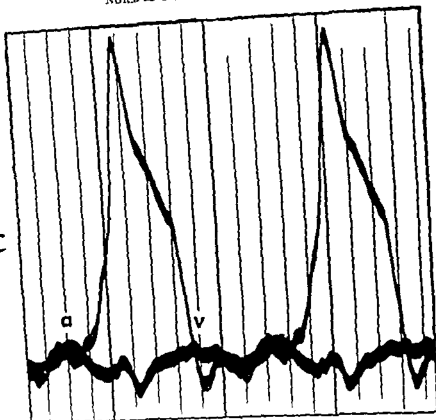


Fig 14—(C) Simultaneous right atrial and right ventricular pulses recorded with the same amplification The right atrial pressure is higher than the ventricular in early diastole identical later

LEFT ATRIUM

The left atrium is easily entered by retrograde arterial catheterization in the dog In man direct puncture appears to be a safer procedure (page 17)

The pressure tracing *of the dog* (Fig 15) shows a rapid often diphasic, presystolic wave (*a wave*) starting about 0 06" before the beginning of the first sound a tall notch during the first sound (*it should be called av*), a deep systolic collapse and a high *v wave*

Left atrial patterns have been recorded in cases of atrial septal defect (or anomalous venous drainage) by Cournand and coworkers[14] and by Nahas et al [18] Direct puncture after thoracotomy allowed Wynn et al [26] to study these pulses in normal subjects Later on Facquet et al [18b] Allison and Linden [27] and Epps and Adler[27] studied the left atrial pulse by transbronchial puncture while Bjoerk[19, 20] and Kent et al [2] studied it by transthoracic puncture

In general the above tracings were recorded with low film speed and

poor amplification, and showed poor detail However, one can gather the following impressions

(a) left atrial tracings are grossly similar to right atrial tracings,

(b) the presystolic rise of pressure occurs *after* that of the right atrium and is taller and sharper,

(c) like the right atrial, left atrial pressure *drops* during ventricular systole

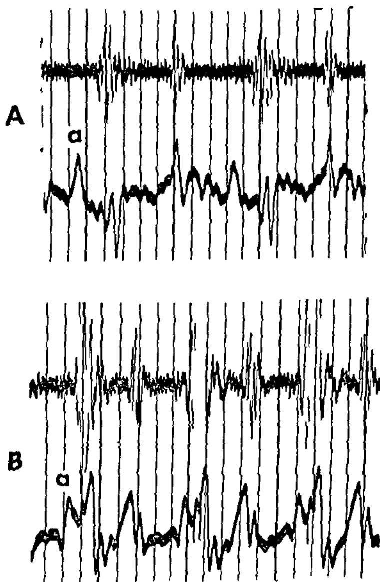


Fig 15—Left atrial patterns in normal dogs

(A) Phonocardiogram and left atrial pattern A = atrial wave

(B) Left atrial pattern in another animal

We have recorded left atrial pressure pulses in several normal dogs (Fig 15) The common features are the following

(a) *The presystolic a wave* of the left atrium is steep and tall and follows the *a wave* of the right atrium. It may start with a small dip

(b) An extremely *small notch* during the first sound This notch which is caused by the closure of the mitral valve should *not* be called c (carotid) but rather *av* (atrioventricular), like the similar wave found in the pressure tracing of the right atrium However it is frequently absent

(c) A *systolic collapse* during ventricular systole

(d) A gradual rise and then a tall *v wave* which coincides with the early diastolic opening of the mitral valve and follows by 0 05 –0 08 the main *vibration of the second* heart sound *

We have not obtained pressure tracings from the left atrium in normal subjects through puncture of the atrium However, we were able to pass the catheter through a patent foramen ovale (or small atrial septal defect) in two infants with no evidence of left to right or right to left interatrial shunt The tracing was similar to the foregoing description Moreover correlation of other types of tracings (esophagocardiograms or left atrial electrokymograms) with pressure tracings of the right atrium confirms the existence of a tall and pointed *a wave* (occurring slightly after the right atrial *a wave* and terminating during the first sound), a systolic collapse, and a well defined, protodiastolic *v wave*

VENTRICLES

Ventricular pressure tracings have been recorded for a long time in animals The plateau like pulse described by Chauveau and Marey[33] in the horse was later recorded also in the dog However, the shorter duration of systole in the latter animal frequently caused this pulse to have a more rounded contour According to a classic description,[1] the typical tracing has a rise during the tension period (first part of first sound), reaches its maximum level at, *or soon after* the end of the first sound, remains level or gently slopes down during systole, then starts to drop rapidly at the beginning of protodiastole (slightly before the second sound) The lowest level is reached some time after the second sound and shortly after the beginning of opening of the A V valves After this, rapid filling begins and a short *phase of* rapid rise occurs, which is soon followed by an even course (Figs 2 and 14C) Certain variations may occur but are not remarkable

In order to exclude the different contours arising from a method different from that employed in man we have catheterized dogs using a method which is identical to that currently used for right heart catheterization in man

* Strangely enough certain authors place this *v* wave in late systole This can be explained only by poor technique or poor timing (the ECG is definitely inadequate for such purpose)

The ventricular tracings of 4 dogs are reproduced in Fig 16 The 2 tracings of Fig 16A and B are similar to classic tracings recorded with older-type manometers The third tracing was recorded with a high sensitivity microphone and an electromanometer It clearly shows two *rapid complexes, one at the beginning, the other at the end of the first* sound It is apparent that they are caused by the rapid swings of the valves first the closure of the tricuspid valve, then the opening of the pulmonic valve These complexes are usually not recorded on account of slower response of the manometer and lesser amplification, this being *particularly true in the case of the left ventricle A conic peak in systole* is definitely atypical The fourth tracing shows a pointed contour which is atypical and can only be explained through artifacts (page 79)

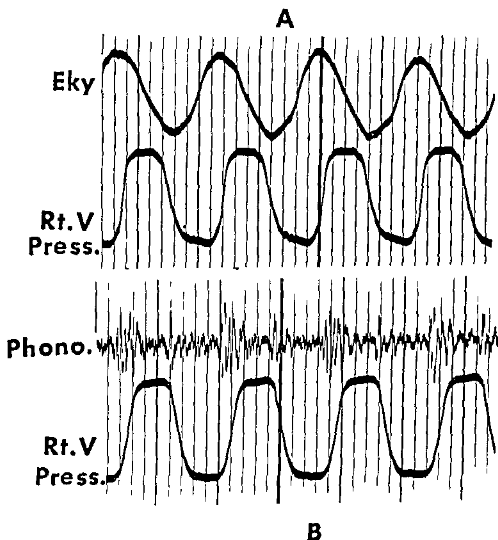


Fig 16—Ventricular patterns of four normal dogs
(A) *Electrokymogram* and pressure tracing of right ventricle
(B C D) Heart sounds and right ventricular pressure

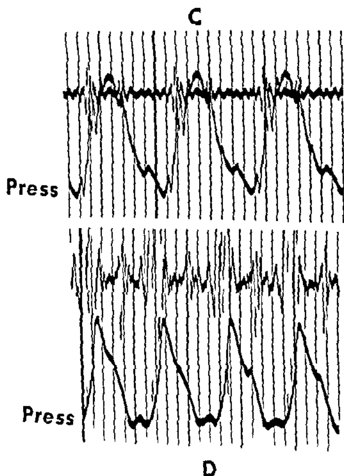


Fig 16—continued

Right Ventricle

Right ventricular tracings from 4 normal persons between the ages of 5 and 22 years are presented in Fig 17 All have a diastolic pressure which is at or near zero (Table I) The only one which corresponds to the theoretically correct profile of right ventricular pressure is the last, a descending slope of the plateau being a common variation The other three tracings present minor abnormalities including slow rise and drop (17 A and B) or conical shape (17 B and C) It is likely that minor artifacts (page 79), not sufficient to alter the levels of pressure, are involved It is important to note that while the top of the plateau like pulse of the ventricle may present different variations, it never shows a progressive rise or a double rise (after the ascending slope), like some of the pathological tracings

Left Ventricle

The tracing of the left ventricle is similar to that of the right There is a rectangular, plateau like wave during the entire systole (Figs 10, 16) Lesser amplification is necessary on account of higher pressure Therefore, the small details of the tracing originating in valvular events are poorly visible or absent The drop in pressure which takes place soon before the second sound shows a rapid descent, the end point of which is at or near zero Early diastole is accompanied by a rapid rise of short duration (rapid inflow), next, a slow slope or a steady course is visible in diastole The highest level of diastolic pressure is identical to the filling pressure of the left atrium

Fig 17—Right ventricular patterns of four normal persons
A = age 20 B = age 22 C = age 14 D = age 5 Upper tracings phonocardiograms

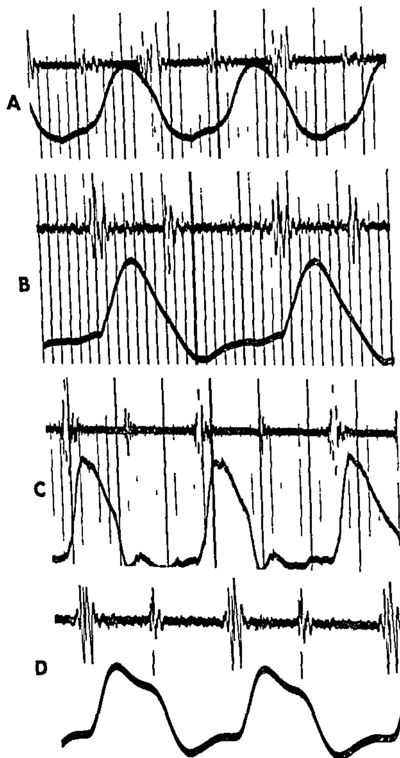


Fig 17

ARTERIES AND VEINS

Aorta, Brachial and Femoral Arteries

The arterial tracings, long recorded in animals, have been registered in man only in the last few years Katz et al [23] have studied the aortic tracing by retrograde arterial catheterization The normal aortic tracing shows the changes of contour which are typical of the *central pulse*[1] the anacrotic depression, the peak, the incisura, and the dicrotic wave Additional waves preceding the rise of the pulse have been explained as caused by presystolic atrial contraction causing a vibration of the aortic valve, and by an early systolic bulging of the aortic leaflets during the tension period

The brachial and femoral pulses, on the other hand, have the smoother contour of the *peripheral pulse* and show no evidence of an anacrotic depression (Fig 18)

Pulmonary Artery

This tracing is typical of an *arterial pulse* with a pattern more similar to that of a *peripheral* than a *central* pulse (Figs 10, 19) The reason lies in the marked distensibility of the pulmonary artery The anacrotic notch is usually not visible, the peak is rounded and falls at about ⅔ of the ejection period, the incisura is deep and rounded and is followed by a high dicrotic wave

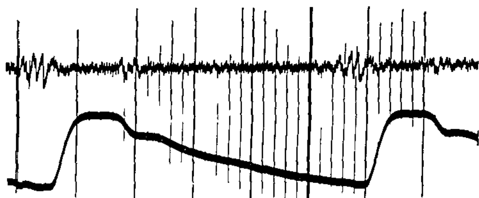


Fig 18—The brachial pulse recorded by arterial catheterization in man Upper tracings phonocardiogram



Fig 19—Pattern of the pulmonary artery Typical normal tracing in a girl of 5

In some tracings, a high, squarish wave or several slow vibrations can be observed in diastole (Fig 47B) These should be considered as artifacts, due to flopping of the catheter after the second sound and the effect of atrial and ventricular movements upon it Careful withdrawal and reinsertion of the catheter may cause the disappearance of these spurious waves

Pulmonary Stems

The pattern of the pressure pulses in the stems of the pulmonary artery is similar to that of the main artery and may be identical with it The peak of the main wave falls slightly later than in the pulmonary artery but still precedes the second sound by about one third of systole

Pulmonary Artery Wedge Tracing

(So called pulmonary "venous" tracing or pulmonary "capillary" tracing) Before describing the pattern of this tracing a word of caution is necessary Technical reasons inherent in the recording of this tracing render it far more variable than any other Several factors should be considered

(a) Possible transmission of pulmonary arterial pulses *around the tip* of the wedged catheter

(b) Possible rapid transmission of left atrial pulses *through open arteriovenous thoroughfares*

(c) *Slow transmission* of left atrial pulses through the minute capillary vessels of the lungs

(d) *Shaking of the catheter* due to movements of the heart (through which it passes) and of the lung (to which it is fixed)

This explains the variability of pattern not only from case to case, but even in the same subject if the catheter is slightly withdrawn and then wedged again

The recording of wedge pressure was described by Hellem Dexter et al[16] in 1948 They correctly attributed the mean pressure recorded by this technique to transmission of left atrial pressure However they recognized that multiple artifacts frequently render the readings incorrect The pressure pulses recorded by *firmly* wedging the catheter into a small pulmonary arterial branch until it occludes the lumen were later studied and described as being similar to those of the left atrium (Hellem et al[18] Lagerloef and Werkoe[19] Soulie et al[41])

It has been stated that because of slow transmission of the waves from the left atrium to the pulmonary arterioles the waves of the "wedge arterial tracing are similar to those of the left atrium but delayed in time If this were so a delay of not less than 0 05" sec should be observed There should be a fixed shifting of both the *a* and the *v* wave The former would fall during or after the first sound (early systole) and the latter

after about one third of diastole or even at mid diastole Actually, as will be shown later, only the *a* wave has a major shift, when present Later, it was suggested that a rapid transmission of pulse could occur through arteriovenous anastomoses * If this is true, it would be understandable why the typical waves are not always present closing of the arteriovenous thoroughfares would prevent any rapid transmission of waves and cause a general damping by the capillary bed Only "mean" pressure would then be reliably recorded

We have recorded pulmonary artery "wedge" tracings in 4 normal dogs, in 4 normal subjects, and in several clinical cases in which the pulses should not have been affected by their disease (small atrial septal defect, arterial hypertension, and moderate pulmonary fibrosis) We have been impressed by the following facts

(1) The pattern is extremely variable and frequently consists of several small waves in systole and diastole
(2) There may be only minimal oscillations of pressure
(3) Exceptional cases present a pattern resembling that described by Soulié et al [11]

When the tracing is typical, the following details can be observed At the time of the *first sound* there is a diphasic oscillation, usually of the positive negative type (Fig 20A), which may be replaced by a mono phasic, acute peak (Fig 36D) This notch is probably caused by a shaking of the catheter because of the double valvular movement which takes place at that time in the right heart Following this vibration, there is a slow and smaller wave which takes place in early systole We have marked this as *a* (Fig 20B) because it is probably caused by a *delayed transmission of the left atrial a wave* This is proven by the fact that the wave takes place *earlier* in cases with a prolonged conduction time (Fig 36C) and is absent in cases with atrial fibrillation During the remaining portion of systole, one can observe either a *systolic collapse* (Fig 20A Fig 36C, D) or a flat line (Fig 20B) With the *second sound* there is a vibration which is usually of the negative positive type (Fig 20B), this is immediately followed by a tall, positive wave coinciding with the opening of the mitral valve which should be called the *v wave* (Fig 20A and B, and Fig 36B and C)

It is interesting to note that while the presystolic wave has a marked delay over the atrial contraction the early diastolic wave has only a minimal delay over the opening of the valve This can be explained by the fact that atrial contraction is weak and causes a wave which moves *backwards* toward the capillary vessels of the lungs while the opening of the A V valve causes a sudden *forward* movement of a long column of blood, from the capillaries to the left atrium

* This concept was advocated by Silber

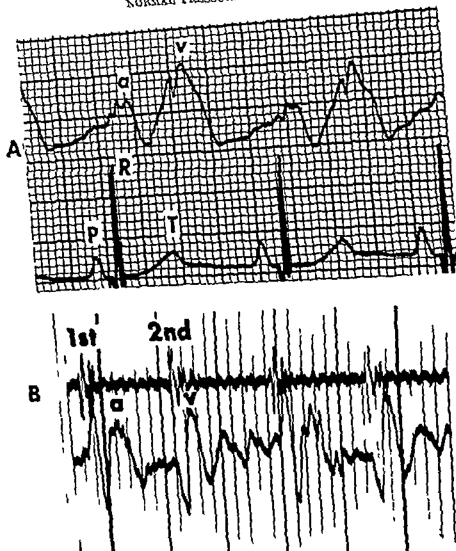


Fig 20—Pulmonary artery wedged tracings in two normal subjects
(a) direct writing apparatus lower tracing = electrocardiogram
(b) photographic recording upper tracing = phonocardiogram

Whenever a recognizable pattern is recorded this may be studied and interpreted otherwise no such study can be made The most undesirable possibility is that of recording a pulse transmitted from the pulmonary artery whenever this happens it can easily be confused with a retrograde systolic wave due to mitral regurgitation

Pulmonary Venous Wedge Tracing

(So called pulmonary arterial "capillary" tracing) This tracing can be recorded by left atrial catheterization done either through the bronchus or the chest wall and by firmly wedging the catheter into a pulmonary

vein A similar tracing can be obtained by right heart catheterization if the catheter passes into the left atrium through an atrial septal defect The observed pattern is that of an arterial pulsation (Gensini et al [42])

CORONARY SINUS TRACING

The technique of catheterization of the coronary sinus was developed by Banfield et al [43] and was studied by Bing et al [44] The patterns of the pressure pulses recorded by wedging the catheter in the coronary sinus, or by having it in the same vessel without obstructing the flow, were studied by Read et al [45] They describe a venous type of tracing if the tip is not obstructing the flow, with no evident *c wave* and a higher pressure than that of the right atrium They also describe a "ventricular" pattern if the catheter is obstructing the flow by occluding the opening

In one case, a child of 5, subsequently recognized as normal, we recorded the coronary sinus pattern with the catheter wedged in the vessel (Fig 21) The tracing had the following characteristics a drop in pressure in late presystole and early systole, a sharp rise in pressure during early systole, and a plateau which continues through the rest of systole *plus* most of diastole

No other known tracing has a similar pattern The form of this tracing may be explained by the systolic squeezing of the capillaries of the ventricular wall plus the well known diastolic flow through the capillary bed



Fig 21—Tracing obtained by wedging the catheter in the coronary sinus Normal 5 yr old girl Upper tracing phonocardiogram

CHAPTER FIVE

Abnormal Pressure Patterns

OUTLINE OF ABNORMAL PATTERNS IN PREVIOUS LITERATURE

Abnormal Right Atrial Patterns

The most common abnormal pattern is that caused by *increased pressure* in the right heart In such cases, the *a wave* becomes tall and lasts longer According to Shepard,[44] the pressure of the *a wave* may reach 10 mm Hg or more Broadening of the wave may be such that it reaches the following *av wave* and may practically fuse with it

Tricuspid insufficiency is revealed by a different pattern which will be described later (page 53)

Atrial fibrillation causes the disappearance of the *a wave* Atrial flutter is revealed by multiple *a waves*

Constrictive pericarditis and other conditions altering diastole are also frequently accompanied by abnormal atrial patterns

Abnormal Left Atrial Patterns

The first abnormal left atrial pressure pulses were published by Munnell and Lam[15] (open chest) as well as by Cournand et al , Lagerloef and Werkoe [40] and Calazel Bing et al [41] (catheter passed through an atrial septal defect) Since then numerous authors have studied this pattern by direct puncture of the atrium either at thoracotomy or with a closed chest (tran bronchial or transthoracic[17b-22]) The importance of such a pattern in rheumatic mitral patients is self evident

Unfortunately inexact terminology incorrect interpretation of some tracings and the division of cases on the basis of either clinical diagnosis or evaluation by the surgeon (both are inadequate for a careful study) render evaluation and comparison of their data extremely difficult

As in the right heart, increased filling pressure of the left atrium is revealed by tall and broad *a waves* and by higher atrial pressure Atrial fibrillation causes the disappearance of the *a wave* Mitral regurgitation is followed by the appearance of an entirely different and new pattern

The studies of Braunwald et al [44] have revealed that a "mitral block" results in a higher level of atrial pressure and, possibly, in an exaggeration of the *a* and *v* waves (Fig 22) In such cases, filling of the atrium will take place more rapidly and will cause an *earlier rise of the tracing in late systole* Any other pattern should be interpreted as being caused by summation of the phenomena due to insufficiency (systolic plateau) with those due to stenosis (high diastolic pressure)

Mitral insufficiency results in the transmission of a typical *plateau pattern* from the left ventricle to the left atrium *The atrial pressure is normal during ventricular diastole* while the pressure pulse during ventricular systole may vary from a steep oblique rise to a squarish plateau, the latter, even though lower, is similar to the pressure pulse of the left ventricle and is an *early plateau* (Fig 23) *

In *mitral stenosis plus insufficiency*, the regurgitant jet must overcome the resistance of the high pressure existing in the left atrium and must pass through a narrow opening These two factors cause a delay in the rise and fall of the wave and a distortion of the pattern, thus resulting in a *late plateau* (Fig 24) The expansion of the left atrium partially absorbs the regurgitant jet in cases with moderate regurgitation, this accounts for the more oblique course of the pressure tracing in comparison with a volume tracing recorded in experimental animals (electrokymogram)[70] (Fig 25A and B) On the other hand, severe regurgitation causes a typical "early plateau" pattern in experimental animals[70] (Fig 25C), as well as in clinical cases

* In cases with nodal rhythm the pressure tracing may require careful study to avoid the rise of pressure due to a late atrial contraction being confused with an early plateau (see pages 75 and 81)

In cases with moderate insufficiency the effect of the normal pull on the A V junction by the left ventricle is still visible It reveals itself as a concave line at the top of the plateau In cases with severe insufficiency not only is there no systolic collapse but the plateau fails to show even this concavity which is the last vestige of the collapse itself

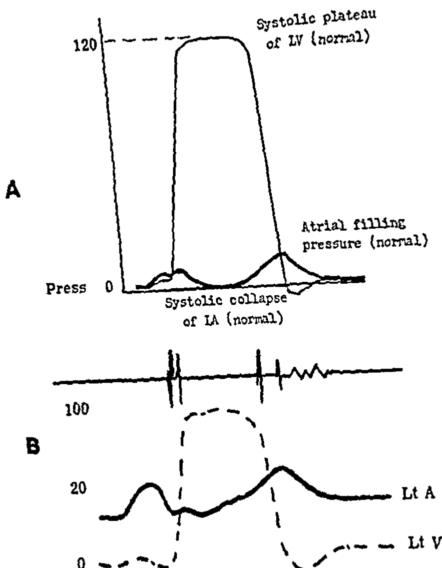


Fig 22—(A) Scheme of normal left ventricular and atrial pressures Full bottom line = [illegible] normal left atrial pressure Thin line = left atrial pressure

(B) Scheme as in (A) in a case of pure mitral stenosis The entire atrial tracing is displaced upwards and there is a pressure gradient between LA and LV

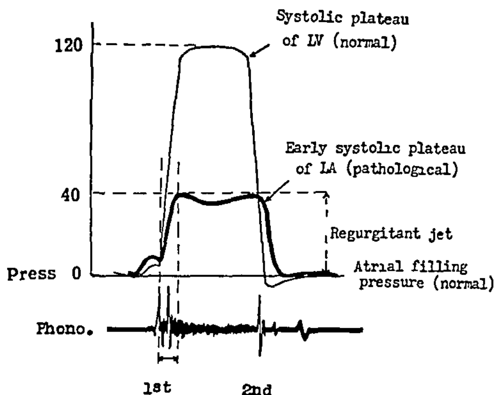


Fig 23—Scheme of left ventricular and atrial pressures in mitral insufficiency There is an early plateau in the left atrium The filling pressure is normal No pressure gradient

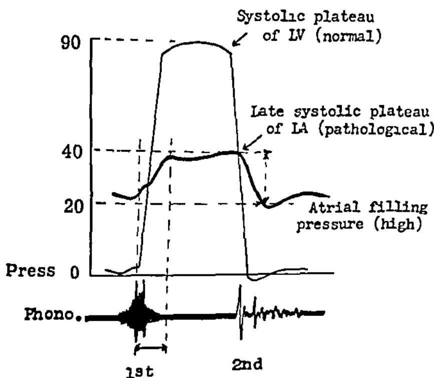


Fig 24—Scheme of left ventricular and atrial pressures in mitral stenosis plus insufficiency There is a late plateau in the left atrium there also is a gradient of pressure between LA and LV

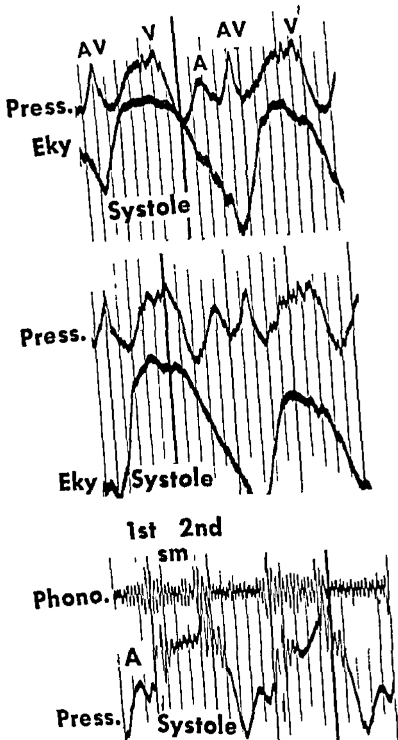


Fig ?—Experimental mitral insufficiency in the dog
(A B) Simultaneous electrokymogram (border motion) and pressure tracing of the left atrium The rise of the tracing occurs earlier in the motion tracing (systolic expansion) than in the pressure tracing
(C) Phonocardiogram and pressure tracing of the left atrium in a more advanced stage Early plateau in the left atrium Systolic murmur

Abnormal Ventricular Patterns

Abnormal ventricular patterns have been the object of systematic experimental investigations by Wiggers.[2] In severe *pericardial effusion*, the initial intraventricular tension rises almost proportionally with intrapericardial pressure but the volume of the ventricles in diastole is decreased due to the compression developed by the fluid. As a result, smaller ventricular pulses are observed and the gradient of the pulse becomes slower.

The immediate effects of *arterial hypertension* are a slightly longer isometric contraction and an ejection period which is so shortened that systole, as a whole, becomes shorter. The pressure rises to a higher peak but a small amount of blood is retained at each beat thus causing a greater initial tension. In a later stage, initial tension is higher, the isometric gradient is steeper, isometric contraction decreases, and the ejection phase becomes more prolonged. *The pattern of the ventricular pulse is* now changed and, instead of a rectangular plateau, there is a *more conical wave with a late peak.*

In *coronary occlusion*, as shown by Orias,[58] the typical changes are a slower rise during the tension period without prolongation of this phase, a shorter period of ejection, and a low rounded peak. The *systolic expansion* of the infarcted area, first demonstrated in animals by Wiggers and Tennant[59] and later confirmed by roentgenkymographic[60] and electrokymographic[61] observations in man, is largely responsible for these changes.

In *pulmonic or aortic stenosis*, the pulse wave of the affected ventricle becomes *higher and more peaked.*[62, 63] *When ejection is severely impeded*, the ventricular contraction becomes nearly isometric, with great rise in pressure and little ejection. While in experimental aortic stenosis the rise in pressure may be tremendous, in experimental pulmonic stenosis the pressure soon falls and the pulse wave is small and conical.

Abnormal Pulmonary Artery Wedge Patterns

A painstaking study was made by several research groups of this tracing in patients with rheumatic valvular lesions. It was hoped to be able to record an entirely different pattern in patients with mitral insufficiency contrasting with cases of mitral stenosis. Interesting observations were made and a new pattern described: *an abnormal, positive wave appearing during the late phase of ventricular systole.* This was probably caused by the mitral regurgitant jet, transmitted to the pulmonary veins (Gorlin et al.,[71] Lagerloef and Werkoe,[40] Soulié et al.[41, 72]). *Heated controversies* have arisen in regard to this pattern. There is now a widespread belief that this pattern is not consistently observed. Moreover, confusion was added by the fact that evaluation of mitral insufficiency

was made through auscultation or digital feeling of a regurgitant jet by the surgeon, both being inadequate methods

RECORDED ABNORMAL PATTERNS

Superior and Inferior Venae Cavae

Several abnormal patterns can be observed

Tall a wave may be found in any case with high venous (and right atrial) pressures (Fig 26A)

Double a wave is a curious phenomenon which still lacks explanation (Fig 26B, C)

Multiple a waves may be observed in atrial flutter

Absence of a waves is observed in atrial fibrillation

Plateau pattern is typical of tricuspid insufficiency

If this dynamic disturbance is functional or is unaccompanied by stenosis, the plateau resembles that of a ventricular tracing, and its rise and fall respectively coincide with the first and second sounds If, on the contrary there also is valvular stenosis, the plateau is a "late plateau," being delayed in its onset and termination (Messer and Sprague), or is irregular, and is taller at the end than at the beginning (Fig 26D)

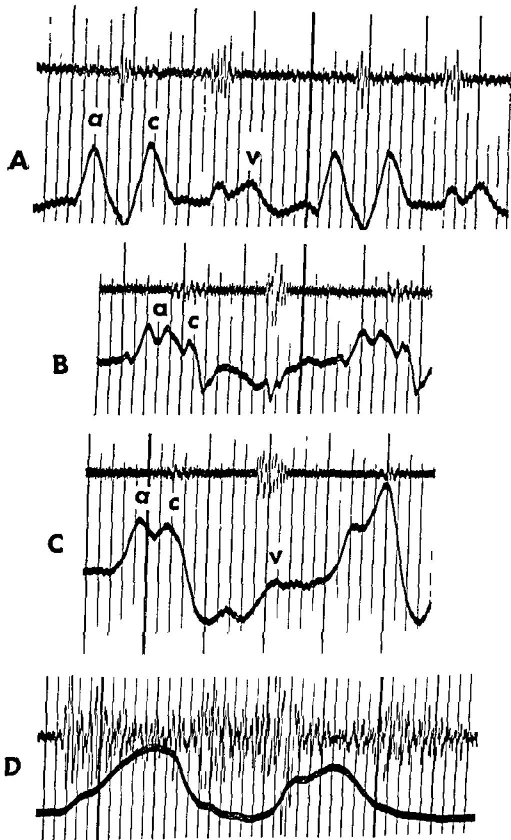


Fig 26—Abnormal caval patterns

(A) *Tall a and c waves Cor pulmonale SVC*

(B) Double *a* wave in Eisenmenger complex *IVC*

(C) A wave followed by tall *c* wave SVC

(D) Plateau pattern Rheumatic heart with mitral and tricuspid lesions IVC

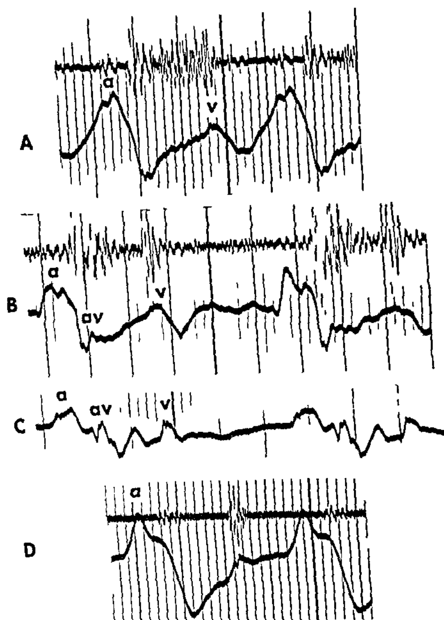


Fig 27—Abnormal right atrial patterns

(A) Tall double peaked *a* waves deep systolic collapse in pulmonic stenosis (press 6 5/0)

(B) Broad *a* waves in pentalogy of Fallot (mean press 6)

(C) Broad double peaked *a* waves in cor pulmonale (press 6/2.5)

(D) Broad *a* waves in Eisenmenger complex (press 7/1)

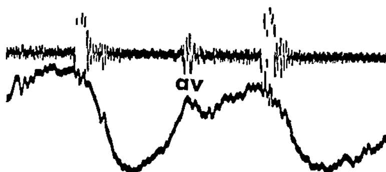


Fig 27 (continued)—Abnormal right atrial patterns

(E) Plateau pattern in tricuspid regurgitation (press 4/—3) The early peak of the plateau is marked as *av* because of the similar mechanism with the *av* wave of the normal tracing

Right Atrial Patterns

The most common abnormal pattern is that of a tall a wave followed by a rapid and deep systolic collapse (Fig 27A) The peak of the wave may be bifid This is typically encountered in pulmonic stenosis with high right ventricular pressure, as already shown by Shepard [52] An extremely tall *a wave occurs* whenever *an atrial contraction takes place* during ventricular systole (Fig 44)

Broad a-wave (nodal or ventricular extrasystoles, nodal tachycardia, complete A-V block) is either tall or normal It is always broad and flat, and may present a *double peak* * We found this pattern in pentalogy of Fallot, cor pulmonale, and Eisenmenger's complex (Fig 27B, C, D) A similar pattern is reported by Shepard[52] in pulmonic stenosis An exact explanation of this curious phenomenon is still lacking

Plateau pattern is typical of tricuspid regurgitation (Fig 27E) In cases of atrial fibrillation, the plateau is particularly apparent on account of the absence of the *a wave* This pattern can be found in cases of rheumatic heart with mitral stenosis and either relative tricuspid insufficiency or organic tricuspid insufficiency It is also typical of Ebstein's syndrome Concomitant tricuspid stenosis (rheumatic cases) is revealed by

(a) delay of the onset and end of the plateau over the heart sounds (late plateau)

(b) increased gradient of pressure in diastole between right atrium and right ventricle (Fig 41)

* Double peaked *a wave* has been found by Gelfand in the jugular tracings of patients with atrial septal defect We have been unable to confirm this finding in most of our cases even though it may be occasionally presented

Left Atrial Patterns

We have, so far, only a few tracings recorded by means of left heart catheterization In general, they conform to the schemes of Figs 22, 23, and 24 However, a special pattern, which is typical of mitral stenosis with only minimal mitral insufficiency (with sinus rhythm), should be described in detail (Fig 27F)

The pressure tracing rises sharply during presystole (*a wave*) and reaches an extremely high level In Fig 27F, this rise starts 0 03" after the peak of P Then as the "mitral block" prevents adequate emptying of the atrium, the pressure further rises during the notch *av* An almost normal systolic collapse follows Then, a tall *v wave* The lowest point of the tracing is reached just prior to presystole and is still high over the zero line The systolic collapse cannot reach even this level because of the high starting point

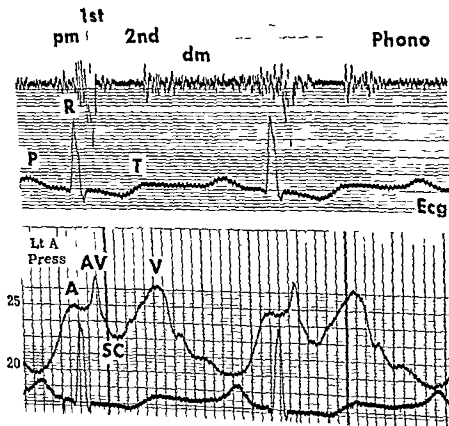


Fig 27 (continued)—Abnormal right atrial patterns

(F) Phonocardiogram and ECG (above) superimposed over a left atrial pressure tracing in a case of pure mitral stenosis

a = presystolic atrial wave
av = wave caused by mitral valve swing
v = early-diastolic wave
sc = systolic collapse

Mitral insufficiency, if present, should reveal itself by a further rise during ventricular systole having a plateau like pattern, possibly obliterating the systolic collapse and causing an upward systolic shift of the pressure tracing (Fig 24) It is important not to confuse the *presystolic* rise of pressure of these cases with the *systolic* plateau of cases also having mitral insufficiency

Right Ventricular Patterns

In our series of cases, we have noted different varieties of the right ventricular patterns which can be considered either normal or borderline (Fig 28) They include

(a) the typical plateau pattern
(b) the conical pattern
(c) the pattern with descending slope

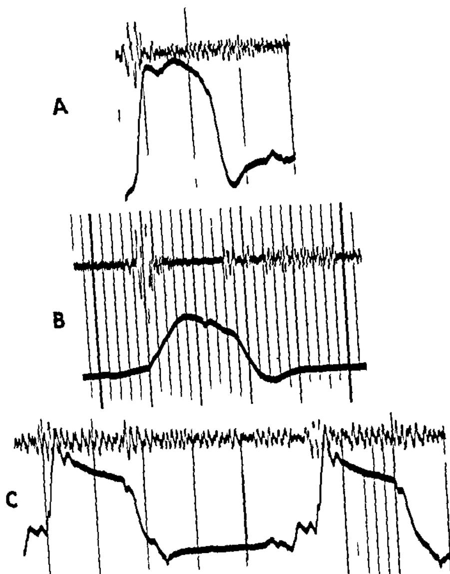


Fig 28—Normal variants of the right ventricular pattern in cardiac patients
(A) Typical pattern (press 30/0)
(B) Conical pattern (press 42/0+7)
(C) Descending slope (press 24/−3−0)

On the other hand, we have found two other patterns which seem to indicate a *definitely abnormal type of contraction* (Fig 29)

(a) the ***double-peaked*** wave

(b) the ***slow ascending*** or ***early-notched*** plateau

These abnormal patterns were found in patients who had a high right ventricular systolic pressure Some of them had an increase of right ventricular *diastolic* pressure, while all presented electrocardiographic evidence of right ventricular hypertrophy and ischemia (strain pattern) It was therefore concluded that these abnormal patterns reflect a condition of "mechanical strain" of the right ventricle

Fig 29—Patterns of ventricular strain
(A) Double peaked wave (press 50/−1+11)
(B) Slowly ascending wave (press 30/+3+6)
(C) Early notched plateau (press 71/+10)
(D) Early notched plateau (press 100/+10+15)

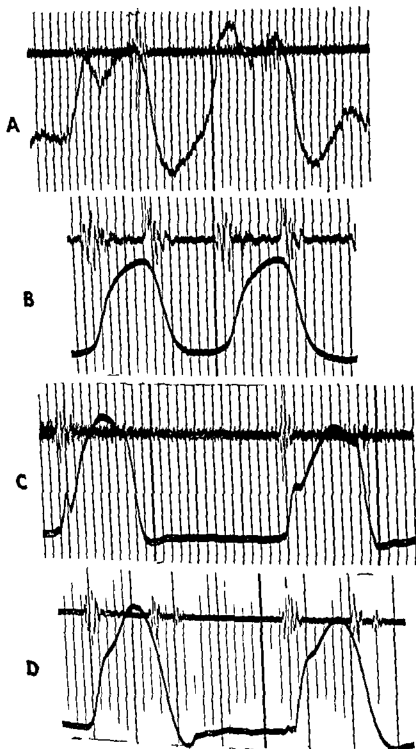


Fig 29

Two other abnormalities of the right ventricular tracing may be observed. One of them consists of a *high diastolic pressure* (9 to 15 mm Hg) due to right heart failure. This high pressure is noticeable at the end of diastole (high filling pressure) while the initial drop may be normal in appearance (Fig 30A). A correspondingly high right atrial pressure is always noted. This high diastolic pressure was described by McMichael[55] and by Harvey.[56] The other abnormality, described by Hansen et al,[57] consists of a *sharp dip in early diastole followed by a flat diastolic course* (Fig 30B). This pattern was originally desscribed in constrictive pericarditis but can be found in other conditions impairing diastole, such as endocardial fibroelastosis or myocardial amyloidosis.[57]

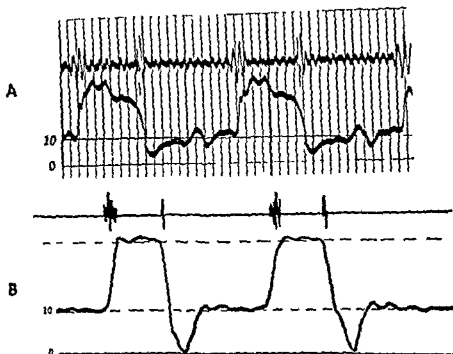


Fig 30—Other abnormal ventricular patterns
(A) High diastolic pressure in a case with initial right ventricular failure
(B) Diastolic dip and flat high diastolic tracing in a case with constrictive pericarditis (scheme after Hansen et al)

Right Infundibular Pattern

This pattern is never encountered unless there is a congenital sub valvular stenosis The latter is caused by a deformity of the ventricular septum which often creates a separate, small, infundibular chamber In such cases, the systolic pressure of the chamber is lower than that of the main right ventricle while the diastolic pressure is lower than that of the pulmonary artery and may be zero A pull back tracing from PA to *infundibulum* and from this chamber to RV clearly shows the differences between the various levels of pressure (Fig 31) The infundibular pattern shows a slowly rising plateau during systole, and may be abruptly terminated by a peak in late systole (Fig 32A and B)

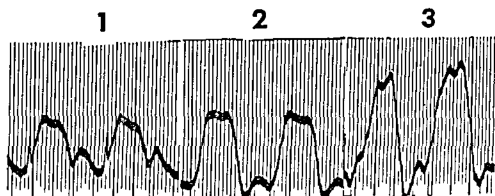


Fig 31—Pull back from PA (1) to first part of infundibulum (2) and to main infundibulum (3) in a case with conical narrowing of last part of infundibulum plus infundibular stenosis of the right ventricle

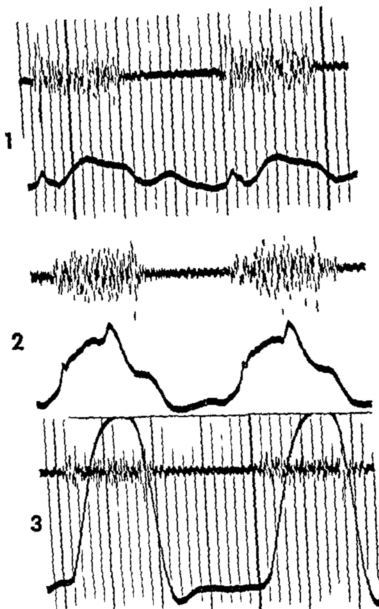


Fig 32—Pressure patterns in the case of Fig 31
(1) Pulmonary artery (press 24/18/16)
(2) Main infundibular chamber (press 35/−2 to +6)
(3) Right ventricle (press 82/−2 to +6)

Pulmonary Artery Patterns

The pulse of the pulmonary artery may present variants due to the *position of the peak* This peak may occur either near the end of the first sound (Fig 33A) or during late systole (Fig 33B) As the position of the peak is determined by the ratio between pulmonary flow and right ventricular output, an early peak logically indicates a free flow A late peak, on the other hand, is probably caused by distal obstruction (arteriolar, capillary, or mitral), relatively weak right ventricle, or both

A second variation is due to the existence of a depression during the ascending branch of the pulse (*anacrotic depression*) (Fig 33C) An anacrotic depression in the aortic pulse is currently interpreted as being due to weakening of the left ventricle in the presence of severe hypertension, or to aortic stenosis It seems logical to explain in the same way the anacrotism of the pulmonary pulse, if artifacts can be ruled out However, severe pulmonic stenosis usually causes a complete transformation of the pattern

The existence of multiple waves in systole, of a high dicrotic wave, or of a negative wave preceding the early systolic rise should be considered as artifacts On the other hand, *absence of the dicrotic wave* (Fig 33B) was noted only in cases of severe pulmonary hypertension

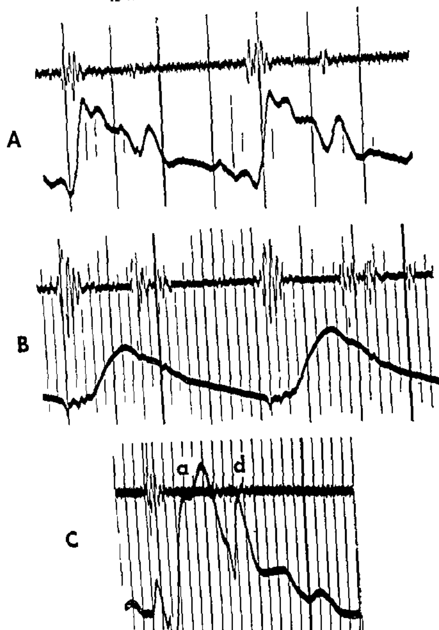


Fig 33—Pulmonary artery pulses
(A) Early peak multiple waves (press 24/9)
(B) Late peak (press 100/55)
(C) Anacrotic depression (a) and high dicrotic wave (d) press 73/39 (See Fig 29 C for corresponding right ventricular pattern)

Severe pulmonic stenosis of the valvular type causes a complete distortion of the pattern (Fig 34) The ascending branch becomes slow and oblique and shows the inscription of several rapid vibrations which are the equivalent of the systolic thrill By analogy with events caused by aortic stenosis, this could be called the "pulmonic shudder" As shown by Fig 35, there may be a late peak coinciding with a loud second sound (obviously transmitted from the aorta) while the second pulmonic sound is extremely weak and delayed

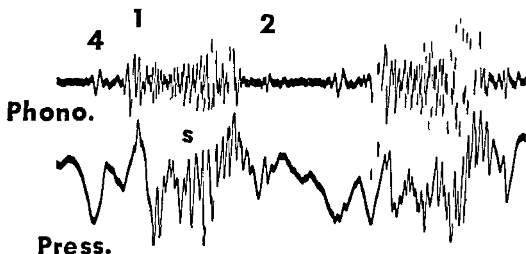


Fig 34—Phonocardiogram and pulmonary arterial pulse (press) in a case of severe pulmonic stenosis Note slow rise and pulmonic ' shudder ' (S)
(2) indicates weak second pulmonic sound
(4) is the fourth (atrial) sound

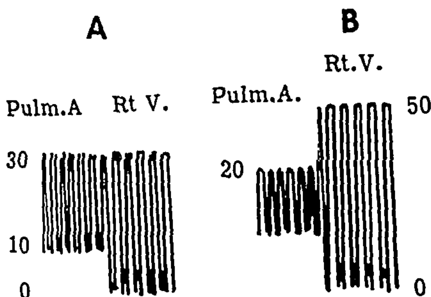


Fig 35—Schemes of gradients of pressure in a pullback from pulmonary artery to right ventricle
(A) Normal—no gradient
(B) Pulmonic stenosis—rise in pressure upon entering the ventricle

Pulmonary Wedge Patterns

As already stated, patterns recorded with the catheter firmly wedged into a small pulmonic branch present wide variations even in normal subjects (page 43) In patients with heart disease, the same variations can be found without diagnostic significance Whenever two waves are observed (one during the first sound, the other soon after the second), the tracing may be considered as typical (Fig 36) In general this pattern corresponds to a level of "wedged" pressure which is within normal limits

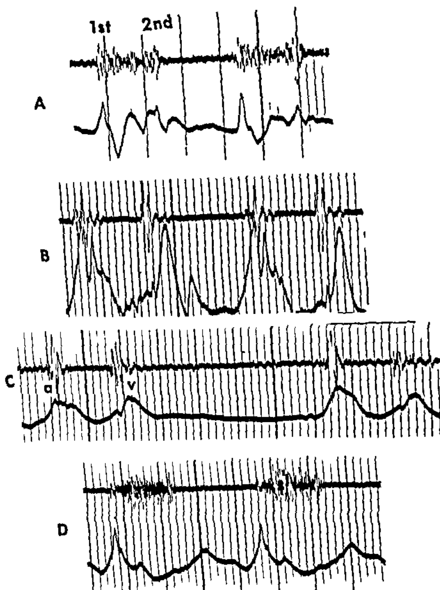


Fig 36—Normal patterns of wedged pulmonary artery pulses in cardiac cases Ventricular systole is between 1st and 2nd sound

(A) Pulmonic stenosis and ventricular septal defect—Mean pressure = 15

(B) Mitral stenosis plus tricuspid insufficiency (record taken one year after mitral valvotomy)—Mean pressure = 13

(C) Mitral stenosis—Mean pressure = 15

(D) Ventricular (and probably atrial) septal defects—Mean pressure = 10

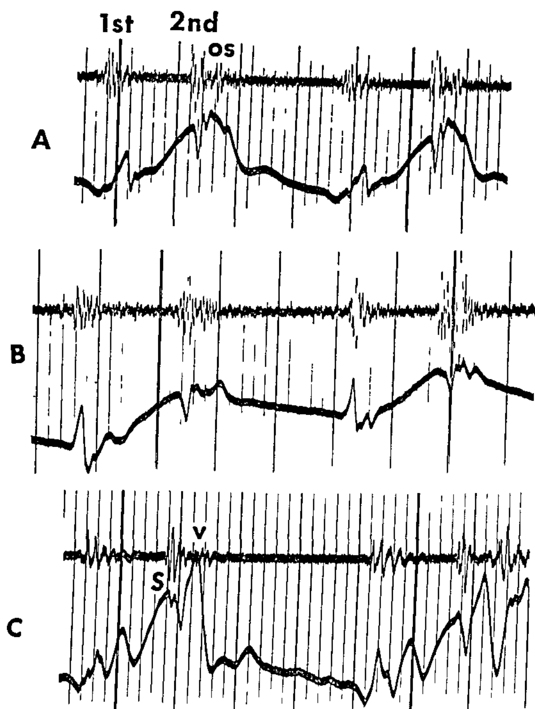


Fig 37—Mitral valve lesions There is an early rise of pressure during ventricular systole
(A) PA wedge = mean 30 (predominant mitral stenosis)
(B) PA wedge = mean 26 (predominant mitral stenosis)
(C) PA wedge = mean 28–30 In this tracing the wave (S) may be revealing some degree of mitral regurgitation

Mitral stenosis with high pulmonary venous and "wedge" pressures (and no appreciable regurgitation) may be accompanied by a pattern of slowly rising pressure (Fig 37A, B) On the other hand, mitral stenosis and regurgitation may be accompanied by *a new wave in late systole* (Fig 37C) As pointed out originally by Gorlin et al [71] Lagerloef and Werkoe [40] and Soulié et al,[41] [72] this wave is due to the transmission across the capillary bed of that regurgitant jet which penetrates the left atrium during ventricular systole because of mitral incompetence Unfortunately, the recording of this wave is inconstant, so that while its presence adds an interesting datum to the various signs of mitral regurgitation its absence does not exclude this defect

Severe variations in the basic level of "wedge" pressure due to respiration are encountered in cases with obstruction of the capillary bed (Fig 38)



Fig 38—Severe respiratory variations of the PA wedge pressures in a case with chronic pulmonary disease

Arrhythmias-Alternans

Ventricular premature contractions are revealed by smaller waves in the tracings of the right ventricle or pulmonary artery (Fig 39) The small wave is nearer the preceding wave and is followed by a pause which may (with ventricular premature contractions) or may not (with atrial or nodal premature contractions) be compensatory

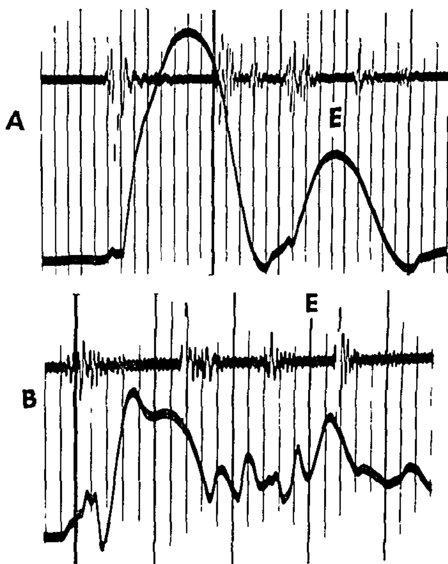


Fig 39—Ventricular premature contractions (F I)
(A) Right ventricular pressure pulses
(B) P A pulses

Alternans is revealed by the regular sequence of a large and a small wave without any irregularity in their sequence (Fig 40) *Left* ventricular alternans indicates damage or functional deterioration of the left ventricular myocardium *Right* ventricular alternans was described by Katz and associates[61] through cardiac catheterization It was subsequently observed by us in two clinical cases (Fig 40) and interpreted as evidence of severe deterioration of the right ventricular function

Nodal rhythm, a disturbance of the rhythm consisting of simultaneous *atrial and ventricular contractions,* may cause typical changes in the *atrial and caval patterns* Mention was already made of them (page 48 and Fig 44) The rise in pressure due to atrial contraction takes place *during part* of ventricular systole and can thus be differentiated from the result of valvular incompetence which would give a plateau wave *during* most or all of systole

Atrial flutter Flutter waves have been described in the tracings of the right atrium right ventricle and pulmonary artery[66] While the atrial waves are certainly due to rapid and coordinated contraction of the atrial walls those of the ventricle and of the pulmonary artery are likely *to* be caused by an unavoidable artifact (shaking of the catheter in the tract passing through the right atrium)

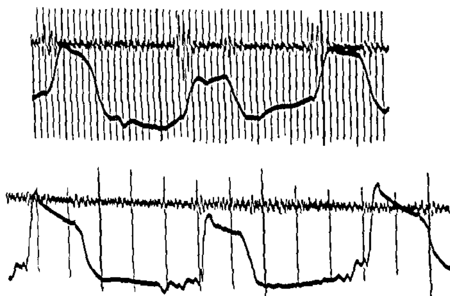


Fig 40—Alternans of the right ventricle in two cases *of* cor pulmonale

Arrhythmias Alternans

Ventricular premature contractions are revealed by smaller waves in the tracings of the right ventricle or pulmonary artery (Fig 39) The small wave is nearer the preceding wave and is followed by a pause which may (with ventricular premature contractions) or may not (with atrial or nodal premature contractions) be compensatory

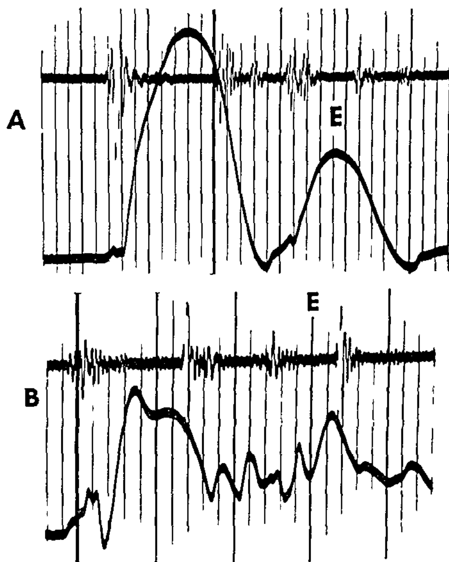


Fig 39—Ventricular premature contractions (L F)
(A) Right ventricular pressure pulses
(B) P A pulses

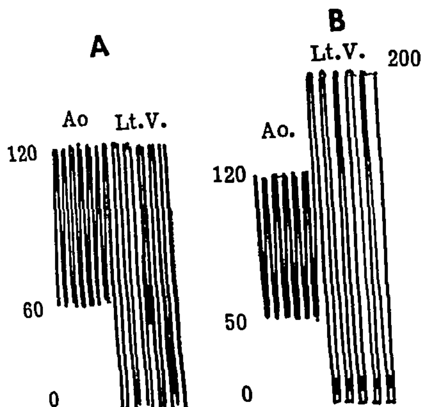


Fig 4?—Gradients of pressure in a pullback from aorta to left ventricle (left heart catheterization)

(A) Normal subject—similar systolic pressure diastolic pressure higher in the aorta

(B) Aortic stenosis—systolic pressure is much higher in the left ventricle than in the aorta

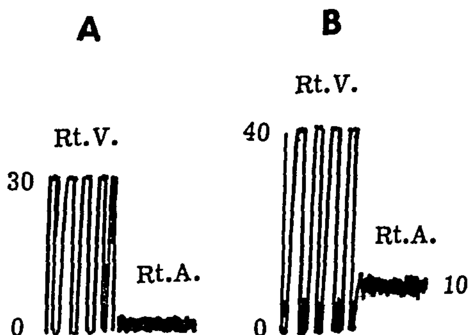


Fig 41—Gradients of pressure between right ventricle and atrium in a pullback from right ventricle

(A) Normal subject—right atrial pressure similar to right ventricular diastolic pressure

(B) Tricuspid stenosis—right atrial pressure higher than right ventricular diastolic pressure

CHAPTER SIX

Artifacts

ARTIFACTS DUE TO FAULTY CONNECTIONS OR RECORDING

There are many factors which can produce artifacts in the patterns of the pressure pulses It is important to recognize them as early as possible during catheterization and to eliminate them immediately, as far as possible If one is not aware of some of these artifacts the tracing of one chamber may be mistaken for that of another

The pressure recording system in cardiac catheterization consists of a radio opaque cardiac catheter connected to a three way stopcock by a Luer lock The second outlet of the stopcock is connected by sterile tubing to a bottle which is provided with a Murphy drip and contains a physiologic solution The third outlet is connected either to a nondistensible lead tube or to a tube made of plastic (or a special rubber) while the other end is connected to the transducer of either a Sanborn manometer, a strain gage or a Hamilton manometer If a Hamilton recording system is used the pressure tracing can be recorded by a photographic device The electric output of the Sanborn manometer or of the strain gage is fed into an amplifier and is recorded by a galvanometer through either a direct writing or a photographic system

From the tip of the catheter to the three way stopcock the tubing and the chamber containing the pressure transducer should be *completely filled* with sterile 5 per cent glucose solution or a physiologic saline solution Because of the physical compressibility of gas any minute air bubble would alter to some extent the pressure tracing *This is one of the most frequent artifacts which may occur in the recording system*

If the air bubble is large there may be no evidence of pressure variations in the tracings If the air bubble is small there will be deflections but the appearance of the pressure tracing will be changed Not only is the timing of curves delayed and the actual pressure level reduced, but the pattern of tracing often becomes indistinct and unusually smooth

When the connections between catheter, 3 way stopcock nondistensible tubing and strain gauge or manometer are not properly secured, leakage tends to occur particularly if the manipulation of the catheter is done in a dark room and the leak is not discovered *Loose connections with slow dripping leakage in the system reduce the pressure* while only

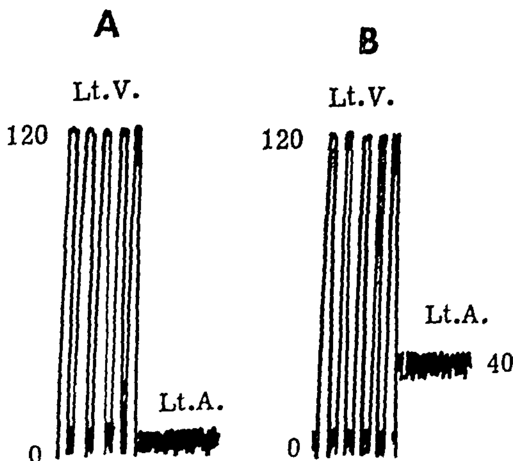


Fig 43—Gradients of pressure in a pullback from left ventricle to left atrium (left heart catheterization)

(A) Normal subject—left atrial pressure similar to left ventricular diastolic pressure

(B) Mitral stenosis—left atrial pressure higher than left ventricular diastolic pressure

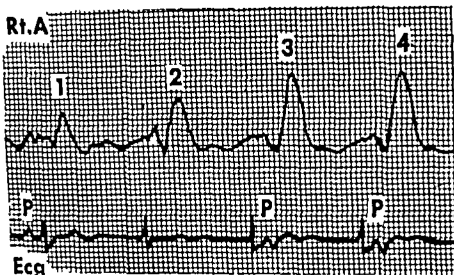


Fig 44—Right atrial patterns in a case of aortic insufficiency Complex 1 has a small positive wave in systole probably caused by minor tricuspid regurgitation Complex 2 corresponding to a nodal rhythm has a higher wave Complexes 3 and 4 corresponding to infranodal beats have giant systolic waves

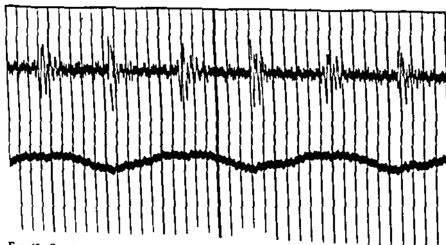


Fig 45—Small pulsations of right atrium due to initial formation of clot in the catheter

slight changes in pattern are observed They are usually recognized with out difficulty while the pressures are recorded Whenever an unusually low pressure is recorded and a smooth pattern observed, checking of the connection and flushing of the system should be carried out at once

Inadequate balancing of the electromanometer due to haste (a "warming up" period of at least one hour is necessary), improper initial balancing, or subsequent imbalance result not only in improper level of pressure but also in abnormal patterns which may vary from inadequate to bizarre The same applies to a strain gage system if the transducer is faulty

Improper calibration of the galvanometer system (overdamping, over shooting) in both the photographic and the direct writing systems may cause incorrect transcription of patterns

Improper application of the writing arm, a too high temperature in the writing system, or a *worn out pen* may also cause faulty tracings

All these artifacts can be avoided by checking the electrocardiograph before preparing for catheterization, following the customary rules for checking such apparatus

ARTIFACTS DUE TO BLOOD CLOTS

In order to prevent fibrin clot formation in the lumen of the catheter, it is necessary to use a continuous drip of heparinized (1000 units per liter) 5 per cent glucose solution in water at a rate of about from 40 to 60 drops per minute, except when blood samples are being collected or pres sure measurements recorded The catheter should be washed with 2 cc of sterile heparinized physiologic saline solution after collection of each blood sample If pressure measurements are carried out for more than a minute or two, such as during a pull back tracing, *a blood clot tends to form near the orifice of the catheter tip* especially when a small (No 5) catheter is used If it is difficult to obtain a blood sample while the catheter tip is in the pulmonary artery or the right atrium and it is still possible to force washed solution fluid under pressure into the catheter, it is likely that *a small fibrin clot has formed at the tip of the catheter, narrowing the open ing with a valve like effect*

It is common to observe, after recording a long pressure tracing, that the graph becomes unusually smooth and the main waves become in distinct or disappear (Fig 45) In such case a small clot has usually formed One can still attempt to obtain a blood sample through gradual suction If however, this is not possible, *it is unwise to force fluid* into the catheter because this might detach a clot from the wall of the latter and cause an embolism Instead, the catheter should be withdrawn and a new catheter be introduced into the vein (unless the essential data have been already obtained)

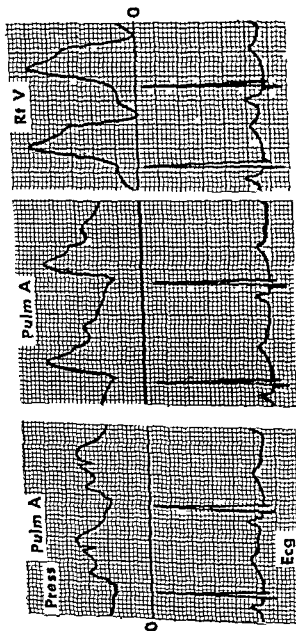


Fig 46—Artifacts in a tracing of pulmonary artery
(A) Right PA
(B) Main PA
(C) Right ventricle

ARTIFACTS DUE TO MOVEMENT OF THE CATHETER

It should be kept in mind that the heart is continuously beating and that the tip of the catheter moves in conjunction with the cardiac action If this movement is prevented, rhythmic occlusion of the tip is far more common The closing and opening of the cardiac valves, the backward and forward movements of the catheter, and the vibrations of the valves, septum or arterial walls, whenever murmurs are produced (Fig 34)—all can be the cause of multiple artifacts

EXAMPLES

The three tracings of Fig 46 were obtained from the same patient while the catheter was in the right pulmonary artery (A), the main pulmonary artery (B), and the right ventricle (C) In Fig 46C, the right ventricular pressure is 25/0/4 5 mm Hg (systolic/early diastolic/end diastolic) The sharp peak may be due to a slight damping of the sensitive manometer In Fig 46B the systolic peak of the main pulmonary artery reaches nearly the same level as that of the systolic pressure of the right ventricle (23 mm Hg), indicating normal size of the pulmonic valve There is a prominent rebound after the end of the T wave

In Fig 46A, the first notch is probably coincident with the first heart sound, the second with the second heart sound, both are probably of valvular origin The systolic deflection, occurring in early systole, is probably due to movement of the catheter and also to impact of the catheter tip against the pulmonary arterial wall

It has been stated that this systolic deflection might be due to a kind of siphon phenomenon due to the velocity of blood flow However the absence of this deflection in Figs 46B and C stands against this suggestion, at least in this case It is obvious that the highest pressure in Fig 46A (17 mm Hg) is the early diastolic rebound (dicrotic wave) and that the systolic pressure of the pulmonary artery cannot be correctly measured Should one record only the deformed pattern of Fig 46A one would consider the possibility of mild pulmonic stenosis on account of a gradient (artifact) between the systolic pressure of the right ventricle and the highest level of the pulse of the pulmonary artery *The use of a catheter with lateral holes near the tip would prevent the occurrence of such an artifact* This is very important because provided that any artifact can be excluded even a gradient of 8 mm Hg between pulmonary artery and right ventricle on a pull back tracing should be accepted as evidence of mild pulmonic stenosis

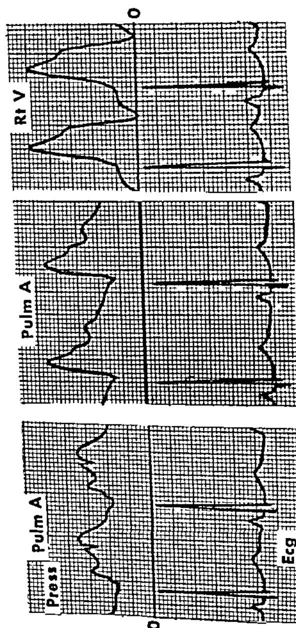


Fig 16—Artifacts in a tracing of pulmonary artery
(A) Right P A
(B) Main P A
(C) Right ventricle

ARTIFACTS DUE TO MOVEMENT OF THE CATHETER

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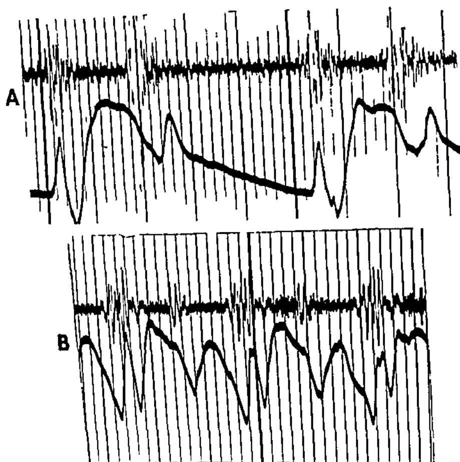


Fig 47—Tracings of pulmonary artery showing artifacts
(A) In a 29 year-old woman
(B) In a 5 year-old child

In Fig 47, an early deflection occurs during the first heart sound It is likely that *flopping of the catheter and its impact on the pulmonary arterial wall during ventricular systole* are distorting the pattern of the tracing of the pulmonary artery

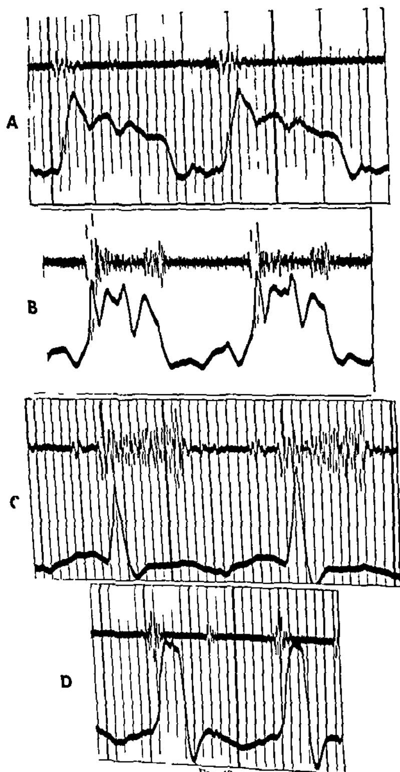


FIG 48

In Fig 48B, the early deflection, occurring immediately after the first component of the first heart sound, may be explained by transient obstruction of the lumen of the catheter by the contracting ventricular wall plus valvular vibrations The late deflection is probably due to transient obstruction of the lumen of the catheter by the contracting ventricular wall

The pressure tracing of Fig 48A was taken while the catheter tip was located in the outflow tract of the right ventricle near the pulmonic valve The early deflection is probably caused by motion of the catheter and valvular vibrations The next deflection is probably due to the impact of the catheter tip against the pulmonary arterial wall or the wall of the right ventricular outflow tract The persisting rise in ventricular pressure during the first half of diastole is probably due to the location of the tip, which is in the pulmonary artery during the first half of ventricular diastole but slips back into the right ventricular outflow tract during the remaining half

In Fig 48C one can see an early systolic rise ending with a sharp peak, followed by an abrupt fall of pressure approaching zero after the first third of ventricular systole It is likely that the orifice of the catheter was obstructed by the contracting ventricular wall during most of ventricular systole

In Fig 48D, after the initial systolic rise, there is a short systolic plateau which begins to fall just before the middle of ventricular systole and approaches zero just after the middle of systole It is possible that this was caused by occlusion of the catheter tip during the last half of ventricular systole

Fig 48—Four tracings of ventricular pressure which are marred by artifacts

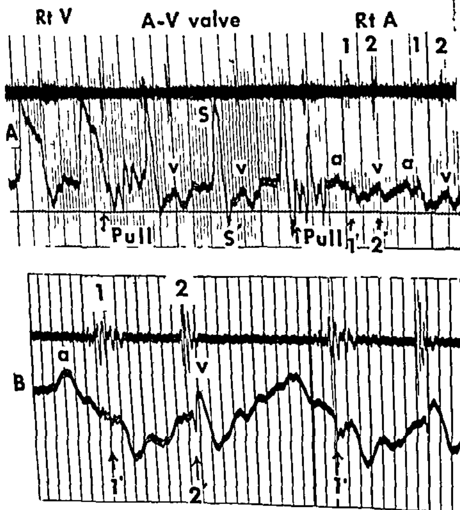


Fig 49—Pullback tracing from right ventricle to right atrium

(A) Low speed large waves *are present* when the tip of the catheter is near the tricuspid valve

(B) High speed catheter in the right atrium some artifacts are still present

During a pull back from the right ventricle into the right atrium, a certain number of deflections are usually recorded, as shown in Figs 8 and 49A The deflection *ss'* is generally accepted as an artifact which is probably due to the impact of the tricuspid leaflets against the catheter tip rather than to the movement of the catheter in itself Subsequently the catheter tip is in the right atrium The small deflections, *1'* and *2'*, are probably valvular in origin and are simultaneous with the heart sounds (Fig 49B)

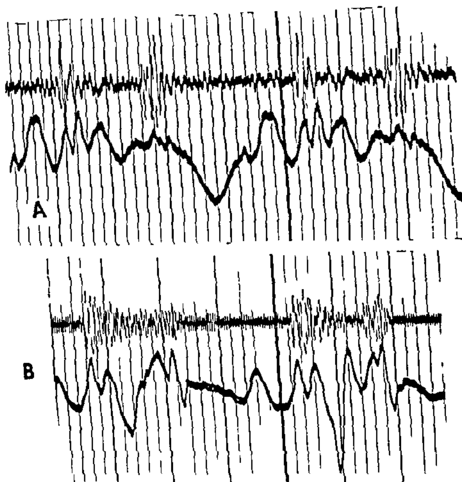


Fig 50—(A) Tracing of the superior vena cava showing artifacts at the time of the first heart sound

(B) Pulmonary arterial wedged tracing revealing multiple artifacts

In Fig 50A, the superior vena caval pressure tracing has an M shaped deflection which is simultaneous with the first heart sound This deflection disappeared in a subsequent tracing recorded after slight rotation and slight pull back of the catheter This M shaped deflection may be due to partial occlusion of the catheter as a result of cardiac movements

It was previously stated that the typical pulmonary arterial "wedge" pressure tracing is seldom recorded (page 43) Theoretically, when the catheter tip is wedged in a small branch of the pulmonary artery, it does not move back and forth simultaneously with the cardiac movements Thus, the pressure tracing recorded should be the same as or similar to those recorded in a pulmonary vein However, it should be emphasized that the catheter passes through the chambers of the right heart and the pulmonary artery, and that the size of the chambers changes from moment to moment as a result of cardiac dynamics The larger the loop made by the catheter within the heart, the greater will be its passive movements and the resulting artifacts (Fig 50B) These should not be considered as actual part of the "wedge" tracing

It is generally agreed that artifacts are commonly found in the pulmonary arterial "wedge" tracing and in many of the pulmonary arterial tracings (Fig 19B and 47) The relationship between artifacts and the dynamic response and susceptibility of the catheter manometer systems caused by movements of the catheter was studied by Wood and coworkers [69]

CHAPTER SEVEN

Formulas Used in Cardiac Catheterization

The calculation of resistance in the pulmonary circulation and of the size of valvular openings presents great interest

By the application of Poiseuille equation

$$\text{resistance} = \frac{\text{pressure gradient}}{\text{rate of flow}},$$

several formulas concerning cardiovascular resistances have been derived for the evaluation of some of the unknown dynamic facts of intracardiac and pulmonary circulations The Poiseuille equation is based on a rigid tube system with continuous flow However, the cardiovascular system has a pulsatile flow and elastic arterial walls Therefore, only approximate data can be obtained from these formulas

RESISTANCE TO BLOOD FLOW

Pulmonary Arteriolar, Total Pulmonary, and Mitral Resistances

It has been suggested that the pulmonary arteriolar resistance or pulmonary vascular resistance proximal to the capillary bed can be measured by formula 1

$$1)\ PAR = \frac{(PA_m - PC_m) \times 1{,}332}{CO} \times 0.06 = \text{dynes seconds cm}^{-5}$$

In this formula PAR is the pulmonary arteriolar resistance PA_m is the mean pressure of the pulmonary artery PC_m is the mean pulmonary capillary pressure (or better mean pulmonary arterial wedge pressure) and CO is cardiac output in liters per minute 0 06 is the reciprocal factor necessary for converting liters per minute into cc per second, 1 332 is the factor for converting mm Hg to dynes per cm²

The *total pulmonary resistance* TPR can be measured by formula 2

$$2)\ TPR = \frac{PA_m \times 1{,}332}{CO} \times 0.06 = \text{dynes seconds cm}^{-5}$$

Here the symbols are the same as in the previous formula and TPR is the total pulmonary resistance

In order to evaluate some of the artifacts which can be produced in a catheter manometer system, certain *in vitro* experiments were made in our laboratory The results are presented in Fig 51

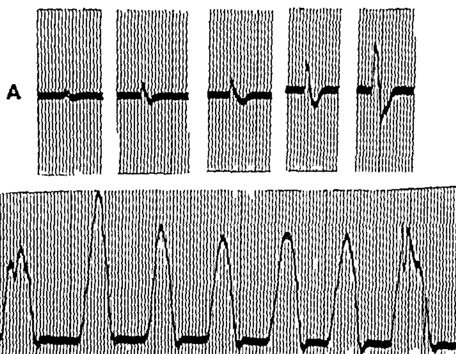


Fig 51—Experiments with a catheter showing artifacts

(A) Open catheter in water longitudinal movements records at various degrees of amplification There are diphasic complexes of proportionally increasing height

(B) Catheter closed by a small rubber balloon Application of pressure on the balloon At the time of the first and last complex the catheter loop was also moved The complexes corresponding to the motion are smaller and more irregular in configuration

Pulmonic stenosis The total resistance of the pulmonic opening can be calculated through formula 6 irrespective of whether there is valvular, infundibular, or a complex type of stenosis

$$6)\ PVR = \frac{(RV_m - PA_m) \times 1{,}332 \times 0.06}{CO} = \text{dynes seconds cm}^{-5}$$

PVR = total resistance to blood flow through the stenotic pulmonic valve
RV_m = right ventricular mean systolic pressure mm Hg
PA_m = pulmonary arterial mean systolic pressure, mm Hg
CO = pulmonary blood flow (liters per min)

The resistance of the infundibulum (PIR) in a complex type of stenosis can be calculated by using formula 7

$$7)\ PIR = \frac{(RV_m - RVI_m) \times 1332 \times 0.06}{CO} = \text{dynes seconds cm}^{-5}$$

RVI_m = Infundibular mean systolic pressure in mm Hg

Likewise the resistance of the stenotic pulmonary valve in a complex type of stenosis can be obtained by formula 8

$$8)\ PPV = \frac{(RVI_m - PA_m) \times 1{,}332 \times 0.06}{CO} = \text{dynes seconds cm}^{-5}$$

The calculated peripheral resistance can be obtained by using formula 9

$$9)\ \text{Peripheral resistance} = \frac{BA_m \times 1332 \times 0.06}{CO} = \text{dynes second cm.}^{-5}$$

Formulas for calculating valvular area are derived by the application of the two well known hydraulic equations a and b

a) $F = CAV$
F = changes in flow rate through the orifice
C = the coefficient of orifice contraction
A = a fixed orifice
V = changes in velocity
b) $V^2 = C\ 2gh$ or $V = C\ \sqrt{2gh}$
V = changes in velocity
h = pressure mm Hg
g = gravity acceleration 980 cm /sec /sec
C = coefficient of velocity (only a certain fraction of pressure is converted to velocity)

The combination of a and b gives the area, A

$$c)\ A = \frac{F}{C \times C_V \sqrt{2gh}} = \frac{F}{C \times 44.5 \sqrt{P_1 - P_2}}\ \text{cm}^2$$

F = flow rate through the orifice
C = discharge coefficient (empirical constant) = 0.7
$\sqrt{2g} = \sqrt{1960} = 44.5$
$P_1 - P_2 = h$ = pressure gradient across the orifice

It has been further suggested that the *resistance of the mitral valve (MVR) can be measured through the difference between formulas 1 and 2* Subtracting the two formulas,[46] one obtains formula 3

$$3)\ MVR = \frac{PC_m \times 1332}{CO} \times 0.06 = \text{dynes seconds cm}^{-5}$$

It is clearly apparent that evaluation of mitral resistance is based entirely on the data of PC_m (or the mean values of pulmonary arterial wedge pressure) which are of doubtful interpretation Moreover, as Burton[46] states, the validity of the formula is based on the assumption that the end diastolic pressure of the left ventricle is zero, a point which is highly questionable, especially in patients with mitral valve disease The possible existence of unknown elements (mitral insufficiency, left ventricular failure, or both) further invalidates these calculations

On the other hand, if the left atrial pressure and the left ventricular diastolic pressure are obtained by means of left heart catheterization, then mitral valvular resistance can be directly measured by formula 4 and the obtained figure is more reliable

$$4)\ MVR = \frac{(LA_{dm} - LV_{dm}) \times 1332 \times 0.06}{CO} = \text{dynes seconds cm}^{-5}$$

Here LA_{dm} is the mean diastolic pressure of the left atrium and LV_{dm} is the mean diastolic pressure of the left ventricle Further deductions from data of catheterization were drawn in order to evaluate the *"elasticity resistance" of the pulmonary vessels*[43] This would be deduced from the data of pulse pressure and flow (cardiac output) The term "elasticity resistance," apparently meaning the reciprocal of distensibility, is then compared to *resistance to flow* for both the pulmonary and the systemic circuits The ratio $\frac{\text{elasticity resistance}}{\text{flow resistance}}$ was found to be 3.5 for the pulmonary circulation and 1.2 for the systemic circulation These data have been criticized[46] and cannot be accepted without further proof

Resistance of Other Stenotic Valves

Tricuspid stenosis By substituting right atrial mean diastolic pressure (RA_{dm}) for the corresponding left atrial data (LA_{dm}) in formula 4, and right ventricular mean diastolic pressure (RV_{dm}) for the corresponding left ventricular data (LV_{dm}) in formula 4 the resistance of the tricuspid valve can be deduced as in formula 5

$$5)\ TVR = \frac{(RA_{dm} - RV_{dm}) \times 1332 \times 0.06}{CO} = \text{dynes seconds cm}^{-5}$$

Work Performed by the Ventricles

The work done by the right ventricle (RVWD) can be calculated by formulas 11 or 11a which are simple and practical Formula 12 is based on the metric system and is more accurate

11) $RVWD = CO\ (PA_m - RA_m) \times 1\,332$ = dynes cm./sec

CO^* = cardiac output cc /sec

PA_m = pulmonary arterial mean systolic pressure mm Hg

RA_m = right atrial mean pressure (may be omitted in order to obtain an approximate value)

1,332 = factor to convert mm Hg to dynes per cm²

Since

1 erg = 1 dyne × cm

1 joule = 10^7 dynes × cm

by substituting joules and omitting RA_m, the formula may be simplified as follows

11a) $$RVWD = \frac{CO \times PA_m \times 1{,}332}{10^7} = \text{joules/sec}$$

A more accurate formulation would be

12) $$RVWD = \frac{(CI \times 1\,055)[PA_m - RA_m) \times 13\,6]}{1\,000} = \text{kgM/min/m}^2$$

kg M (meter)/min /m² (square meters of body surface)

CI^* = cardiac index in liters/min /m²

1 055 = specific gravity of the blood

13 6 = specific weight of mercury

It can be further simplified by omitting RA_m

12a) $$RVWD = \frac{(CI \times 1\,055)\ PA_m \times 13\,6}{1\,000} = \text{kgM/min/m}^2$$

By substituting brachial or femoral mean systolic pressure (BA_m) for PA_m and LA_m for RA_m the work done by the left ventricle (LV) can be also evaluated

The calculation of the amount of blood flowing through shunts requires knowledge of several data including oxygen consumption by the patient and the oxygen contents and pressures of the various cardiovascular chambers and of the peripheral blood For this reason, even though this study was particularly concerned with the pressures and pulses of the various chambers the following pages will refer to oxygen determin

* Cardiac output = pulmonary output and cardiac index = pulmonary index in the absence of shunts

Mitral Valve Area

An attempt to measure mitral valve area (MV_A) was made by Gorlin and Dexter[49, 50] through a modification of hydraulic equation c which gives formula 10

$$10)\ MV_A = \frac{MV_F}{31\sqrt{PC - 5}}$$

This formula is based on the ratio of mitral valvular flow to pulmonary "capillary" (or wedge) pressure

MF_F indicates mitral valvular rate of flow in cc per second and is obtained by dividing the *cardiac output in cc per minute* by the duration of the sum of the diastolic periods (in seconds) The latter is obtained by measuring the diastolic filling per beat and multiplying by the heart rate per minute

Simultaneous pressure tracings of the left ventricle and atrium showed that filling of the left ventricle through a stenotic mitral valve may last through the early part of the ventricular tension period Therefore, duration of diastolic filling can be measured with accuracy only by either phonocardiograms or left heart catheterization

The number 31 used in formula 10 is an empirical constant which is supposed to correct for anomalies of discharge through the orifice and errors in calculating the diastolic filling period, and convert mm Hg to cm H_2O The figure 5 used in formula 10 is acceptable as the estimated level of left ventricular mean diastolic pressure

Those interested in the formulas for calculating the area of the pulmonary, tricuspid, and aortic valves, and of the diameter of a patent ductus arteriosus, or the areas of interatrial or interventricular septal defects, can find data in a publication by the Gorlins [49]

The authors agree with various criticisms which were raised against these attempts to apply well known formulas which should be based on reliable data and simple hydraulic phenomena, to the diseased heart where unknown variables are necessarily overlooked (including mitral regurgitation, aortic defects, left ventricular failure) and somewhat unreliable data are used They agree with Burton[46] who states "The measurement of the elevation of pulmonary pressures in mitral stenosis is a valuable one and will serve as an index of the severity of the narrowing of the valve Why spoil such a scientific measurement by building upon it such a house of cards, the deceptive facade of which may well mislead the trusting clinician into buying it?' Mitral valve estimation through left heart catheterization, even though not devoid of possible errors, is definitely more reliable

In *isolated atrial septal defect* with left to right shunt, the data obtained through catheterization are more reliable than in isolated ventricular septal defect or patent ductus arteriosus, because the blood obtained from the pulmonary artery is already thoroughly mixed

If there is evidence of double left to right shunt through both an atrial and a ventricular septal defect the calculated amounts for the single shunts are not too reliable However, the total amount of left to right shunt is as reliable as in an isolated ventricular septal defect

In the presence of a large left to right shunt through a patent ductus arteriosus the presence of a high ventricular septal defect can be ruled out only with difficulty because one cannot exclude penetration of shunted blood with high oxygen content from the pulmonary artery into the right ventricle because of pulmonic insufficiency

Calculation of the severity of a pure right to left shunt or a bidirectional shunt in atrial septal defects is easily made

FORMULAS FOR CALCULATION OF A SHUNT*

Absence of Shunts

In the *absence of shunts* according to Fick,[78] the cardiac output (or estimated systemic blood flow SBF which equals pulmonary arterial blood flow PBF) is calculated by dividing the oxygen consumption by the difference between arterial and mixed venous bloods (pulmonary arterial sample)

1) $SBF = PBF = \frac{O_2 \times 100}{BA - PA} = cc/min$

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* The formulas for shunts use the following *symbols* and abbreviations

O_2 = O_2 consumption cc /min

SBF = systemic blood flow cc /min

PBF = pulmonary blood flow cc /min

EPBF = effective pulmonary blood flow cc /min

$PI_{PA, RV}$ = pulmonary insufficiency cc /min

SIVC = O_2 *content of both vena cava* vol per cent (SVC × 0 40 + IVC × 0 60)

RA_s = O content of average right *atrium* vol per cent

RV = O_2 content of average right ventricle vol per cent

$RV_{M, H}$ = O_2 content of average of mid and high right ventricle vol *per cent*

PA = O content of pulmonary artery vol per cent

PA = O_2 content of average left and right *pulmonary* arteries vol per cent (in patent ductus arteriosus)

LA = O content *of left* atrium vol per cent

BA = O_2 content of brachial artery *vol per cent*

PV = O content of pulmonary vein vol per cent (*assumed to* be 95 per cent saturated)

P_{RA} = right atrial pressure mm Hg

P_{RV} = right ventricular pressure mm Hg

P_P = pulmonary arterial pressure mm Hg

P_B = brachial arterial pressure mm Hg

Y = left to right shunt (YA = atrial level YV = ventricular level)

Z = right to left shunt (ZA = atrial level ZV = ventricular level)

ation data Thus most of the formulas needed in cardiac catheterization will be included in a single, brief chapter

Intracardiac Shunts

The calculation of intracardiac shunts was established soon after right heart catheterization was introduced as a diagnostic procedure Much of the following outline is universally accepted

(1) The oxygen contents of the superior and inferior venae cavae usually differ by one to three volumes per cent The blood sample of the inferior vena cava should be taken when the tip of the catheter is just below *the diaphragm If this tip advances to the orifice of the hepatic* veins (which usually carry blood with a lower oxygen content) while the sample is collected, then the oxygen content of the sample will be lower *than it should (hepatic blood instead of mixed lower caval blood) If the* tip is below the orifice of the hepatic veins, then the oxygen content of the sample is higher

(2) The inferior vena cava carries about 55 to 60 per cent of the total amount of blood into the right atrium while the superior vena cava carries 40 to 45

(3) The coronary sinus carries about 5 per cent of the total output, consisting of blood with a very low oxygen content (3 to 4 volumes per cent) This blood will be mixed in the lower part of the right atrium and, more thoroughly, in the right ventricle

(4) "Mixed venous blood" is more thoroughly mixed in the pulmonary artery *than in the right atrium or ventricle of patients having no evidence* of shunt

(5) The following data are considered by us to be evidence of left to right shunt between the atria

(a) The average oxygen content of the right atrium should be at least 1 5 volumes *per cent higher than the average of the blood samples of the two venae cavae*

(b) The oxygen content of a single right atrial blood sample should be at least 3 volumes per cent higher than the average of the blood samples of the two venae cavae

(c) The oxygen content of two or more right atrial blood samples should be at least 4 volumes per cent higher than that of either caval sample

(6) In the absence of primary alveolar changes interfering with gas diffusion, a 95 per cent oxygen saturation in the blood of the pulmonary veins or left atrium is considered normal Several factors prevent the theoretical full saturation (100 per cent)

(7) In spite of conflicting statements, we accept pulmonary venous return as identical with pulmonary arterial flow

7) $Y_{PA} = PBF - SBF =$ cc./min.

7a) $Y_{PA} = SBF \dfrac{(PA_{ve} - RA_{[illegible]})}{(BA - PA_{ve})} =$ cc./min.

The pulmonary blood flow (PBF) of formula 7 should be calculated by formula 8 from the average of samples collected from both the left and the right pulmonary arteries It is preferable to obtain 2 samples of blood from each stem of the pulmonary artery (4 in all)

8) $PBF = \dfrac{O_2 \times 100}{(BA - PA_{[illegible]})} =$ cc./min

If only one pulmonary random blood sample is obtained near the orifice of the patent ductus, it may contain a much higher oxygen content, and the pulmonary blood flow thus calculated may become as high as 40 liters/min This is obviously incorrect

In formula 7a, the use of RA_{ve} instead of $RV_{[illegible]}$ is based on the fact that there may be pulmonary regurgitation which would increase the oxygen content of the right ventricular samples especially in the upper part of the ventricle

With both atrial and ventricular defect

$RA_{[illegible]} > SIVC$ (by more than 1 5 vol per cent) while
$RV > RA_{[illegible]}$ (by more than 1 0 vol per cent) and
$RV = PA$

Y_A the left to right shunt through the atrial defect can be calculated by formula 5b Y_V, the left to right shunt through the ventricular defect, can be calculated by formula 9 or 9a

9) $Y_V = PBF - (SBF + Y_A) =$ cc./min

9a) $Y_V = \dfrac{(SBF + Y_A)(PA - RA_{[illegible]})}{(BA - PA)} =$ cc./min

Y_{A+V} the total left to right shunt can be obtained by formulas 10 and 10a

10) $Y_{A+V} = PBF - SBF =$ cc./min.

10a) $Y_{A+V} = \dfrac{SBF(PA - SIVC)}{(BA - PA)} =$ cc./min

With ventricular septal defect and patent ductus arteriosus When both are present the following will be true

$SIVC = RA_{[illegible]}$
$RV_{[illegible]} > RA_{[illegible]}$ (by more than 1 0 vol per cent), and
$PA > RV$ (by more than 1 5 vol per cent)

Y_V the left to right shunt through the ventricular defect, can be calculated by formula 6b

Left to Right Shunts

In the right atrium *Anomalous pulmonary venous return into the right atrium, or atrial septal defect with left to right shunt,* is suspected when $RA_{avg} > SIVC$ (by 1.5 vol per cent or more) and $RA_{avg} = PA$

Anomalous pulmonary venous return into either vena cava is suspected when the difference in O_2 content is more than 4.5 vol per cent between inferior and superior venae cavae

The systemic blood flow in the first instance is calculated through *formula 2,* and the pulmonary flow through *formula 3*

$$2)\ SBF = \frac{O_2 \times 100}{BA - SIVC} = cc/min$$

$$3)\ PBF = \frac{O_2 \times 100}{BA - PA} = cc/min$$

The left to right shunt can be easily obtained by subtracting systemic blood flow from pulmonary blood flow as in formula 4

$$4)\ Y_A = PBF - SBF = cc/min$$

Also, the left to right shunt can be estimated from the total O_2 content of blood passing through the right atrium in one minute as in formula 5

$$5)\ SBF \times SIVC + Y_A \times BA = SBF \times RA_{avg} + Y_A \times RA_{avg}$$

This equation can be simplified as follows

$$5a)\ Y_A(BA - RA_{avg}) = SBF(RA_{avg} - SIVC)\ \text{Next}$$

$$5b)\ Y_A = \frac{SBF(RA_{avg} - SIVC)}{(BA - RA_{avg})} = cc/min$$

In the right ventricle Left to right shunt between ventricles is presumed if $SIVC = RA_{avg}$ and $RV_{avg} = PA$, but $RV_{avg} > RA_{avg}$ (by more than 1.0 vol per cent)

The left to right shunt can be estimated by using either formulas 6 or 7

$$6)\ Y_V = PBF - SBF = cc/min$$

$$6a)\ Y_V = \frac{SBF(PA - RA_{avg})}{(BA - PA)} = 6b)\ \frac{SBF(RV_{avg} - RA_{avg})}{(BA - RV_{avg})} = cc/min$$

Between aorta and pulmonary artery Left to right shunt between the larger arteries is presumed if $SIVC = RA_{avg} = RV_{avg}$, but $PA_{avg} > RV$ (by more than 1.0 vol per cent)

A left to right shunt through a patent ductus arteriosus or any other aorto pulmonary communication can be estimated by formulas 7 or 7a

left atrium in one minute It is assumed that the pulmonary venous blood is 95 per cent saturated with oxygen

14) $PBF \times BA + Z \times BA = PBF \times LA + Z \times PA$

14a) $Z (BA - PA) = PBF (LA - BA)$

14b) $Z = \frac{PBF(LA - BA)}{(BA - PA)} = cc/min$

Ventricular septal defect with or without pulmonic stenosis (tetralogy of Fallot or Eisenmenger complex)

If RA_s = RV = PA there is no left-to-right shunt

If P_{RA} mean is not elevated (less than 6 mm Hg) there is probably no right to left shunt through an additional atrial septal defect

If BA sat < 92 per cent there is right to left shunt

The systemic blood flow pulmonary blood flow and right to left shunt can be likewise calculated by formulas **13, 13a** and **13b** or **14**

Patent ductus arteriosus with pulmonic hypertension The calculations of the right to left shunt can only be based on certain assumptions (1) The shunt takes place from the pulmonary artery to the aorta at a point (*ductus*) which is below the opening of the left carotid artery and above that of the left subclavian artery (2) A minor portion of the shunted blood goes back to the aortic arch and penetrates the innominate artery and the two carotids while a major *portion is* distributed between left subclavian and descending *aorta* This second assumption although used by some is definitely unwarranted because, in some cases, no evidence of low oxygen saturation can be found *in either* the left arm or the left earlobe Therefore as we have no accurate way of ascertaining this distribution of blood accurate mathematical calculation of the amount of shunt is impossible

Bidirectional Shunts (Left to Right plus Right to Left Shunts)

Atrial septal defect with or without pulmonic stenosis The calculations are based on the data of the total O *content* which contributes to the mixing in the left and right atria in one *minute* It is assumed that RA_s = RV = PA and that LA = 95 per cent O_2 *saturation* Formulas **15** and **16** are used

15) $PBF = \frac{O \times 100}{LA - PA}$ cc/min.

16) $SBF = \frac{O_2 \times 100}{BA - SVC}$ cc/min

In the *right atrium* the amount of blood which contributes to the mixing in one minute is calculated by formulas **17, 17a 17b** and **17c**

Y_{PA}, the left to right shunt through a patent ductus, can be calculated by *formulas 11 and 11a (formula 11 is preferred for its simplicity)*

11) $Y_{PA} = PBF - (SBF + Y_V) = $ cc./min.

11a) $Y_{PA} = \dfrac{(SBF + Y_V)(PA_{avg} - RA_{avg})}{BA - PA_{avg}} = $ cc./min

In cases of large left to right shunt due to patent ductus *arteriosus* (without any ventricular septal defect), one may find that the oxygen content of the right ventricular sample (especially if obtained from the outflow tract) is somewhat higher (1 0 vol per cent) than that of the samples of the right atrium and of the inflow tract (or lower part) of the right ventricle In such cases we use RA_{avg} instead of RV_{avg}

At times it is difficult to decide whether or not there is an additional high ventricular defect or pulmonic insufficiency

Pulmonary Insufficiency If *pulmonary insufficiency* is suspected in a case *with patent ductus arteriosus*, the amount of regurgitation can also be calculated, but the result obtained from the average of high and *mid right ventricular samples gives only a rough estimate (formula 12)*

12) $PI_{PA-RV} = \dfrac{SBF(RV_{H+M} - RA_{avg}) \times 100}{(PA_{avg} - RV_{H+M})} = $ cc./min

Right to Left Shunts

Atrial septal defect with or without pulmonic stenosis This shunt can be calculated by comparing the pressures of the right ventricle and brachial artery, simultaneously recorded The O_2 saturation of BA is below *92%*

If $P_{RVS} < P_{BAS}$ there is no right to left shunt provided that overriding of the aorta is ruled out by angiocradiography

If $P_{RVS} > P_{PAS}$ pulmonic stenosis is present but still there is no shunt

If $P_{RAM} = P_{RVD}$ and is slightly elevated (6 to 10 mm Hg) and moreover, BA sat < 92 per cent and $RA_{avg} = RV_{avg} = PA_{avg}$ then there is evidence of a right to left shunt through an atrial septal defect Such a shunt can be calculated from formula 13b derived from formulas 13 and 13a

13) $SBF = \dfrac{O_2 \times 100}{BA - PA} = $ cc./min

13a) $PBF = \dfrac{O_2 \times 100}{LA - PA} = $ cc./min

13b) $Z = SBF - PBF = $ cc./min

In formulas 14 to 14b the calculation of right to left shunt is based on the total O_2 content of the blood which contributes to the mixing in the

Formula 22 is useful in the former cases and 23 in the latter

22) $EPBF = \frac{O_2 \times 100}{PV - SIVC} = cc./min.$

23) $EPBF = \frac{O_2 \times 100}{LA - RA_{4,5}} = cc./min.$

Pulmonic stenosis, especially in cases of tetralogy of Fallot, may be accompanied by large collateral (bronchial) circulation or by a patent ductus arteriosus In such cases total pulmonary blood flow or pulmonary capillary blood flow is larger than the main pulmonary arterial blood flow The former can only be calculated by indirect means, and the result is only a rough estimate [14]

17) $(SBF - Z)SIVC + Y \times LA = (SBF - Z + Y) \times PA$

17a) $SBF \times SIVC - Z \times SIVC + Y \times LA = SBF \times PA - Z \times PA + Y \times PA$

17b) $Y(LA - PA) = SBF(PA - SIVC) + Z(SIVC - PA)$

17c) $Y = \dfrac{SBF(PA - SIVC) + Z(SIVC - PA)}{LA - PA}$

In the *left atrium*, the amount of blood which contributes to the mixing in one minute is calculated by formulas 18, 18a, 18b and 18c

18) $(PBF - Y)LA + Z \times SIVC = (PBF - Y + Z) \times BA$

18a) $PBF \times LA - Y \times LA + Z \times SIVC = PBF \times BA - Y \times BA + Z \times BA$

18b) $Z\,(BA - SIVC) = PBF(LA - BA) + Y\,(BA - LA)$

18c) $Z = \dfrac{PBF(LA - BA) + Y(BA - LA)}{(BA - SIVC)}$

Substitute **18c** into **17c** and formula **19** is derived

19) $Y = \dfrac{SBF(PA - SIVC) + \left(\dfrac{PBF(LA - BA) + Y(BA - LA)}{BA - SIVC}\right)(SIVC - PA)}{(LA - PA)}$

Knowing pulmonary flow, effective pulmonary flow, and systemic flow, *we can use Bing's formulas (20 and 21) for calculating right to left and* left to right shunts The "effective pulmonary flow" EPBF will be *described below*

20) $Y = PBF - EPBF =$ cc./min.

21) $Z = SBF - EPBF =$ cc./min

Eisenmenger complex In this case $SIVC = RA_{avg}$ and $RV_{avg} > RA_{avg}$, but $RV_{avg} = PA$

As in atrial septal defect with bidirectional shunt we can use formulas **15**, *16*, **17c**, **18c**, *19* or **20** and **21** in order to calculate the left to right (Y) and right to left (Z) shunts

In order to obtain a more reliable result, it is advisable to substitute the SIVC for RA_{avg}

The calculation of the "effective pulmonary blood flow" EPBF was advocated by Bing,[76] and it is defined as the amount of mixed venous blood which, having returned to the heart from the systemic circulation, eventually reaches the pulmonary capillaries *Normally*, *the effective* pulmonary blood flow equals pulmonary arterial flow except when bidirectional shunts are present—*as in some cases of atrial septal defect* complicated by pulmonic stenosis, and in cases of the Eisenmenger complex

TABLE IV *Catheterization in Complex Congenital Malformations*

Dis as	P res							Oth ig fi t d t f th t i zati	Oth test hi h m y b f i	Rem k
	RA	RV	PA	PA wedg	LA	LV	AO			
Tetralogy of Fallot	Normal	Markedly elevated	Normal or low	Normal	Normal	*Normal*	Normal	Low O_2 saturation of arterial blood (esp after exertion)	Angiocardiography ECG chest films Phono	
Eisenmenger Complex	Normal	Markedly *elevated* (similar to LV)	Markedly elevated (systolic pressure is as high *as systolic* of RV)	Normal	Normal	Normal	Normal	Low O_2 saturation of *arterial* blood (esp after exertion)	Angiocardiography LCG chest films Phono	
Complete Transposition of Large Vessels	Normal or slightly elevated	Elevated (systolic pressure is regulated by resistance in greater circulation)	Systolic is as high as *that of* LV	Normal	Normal	Low or Normal (systolic can be as high as that of RV)	Systolic is as high as that of RV	Low O_2 saturation of arterial blood	Angiocardiography LCG chest films	An atrial ventricular or ductal opening is *usually present* with equal and bidirectional shunts
Tricuspid Atresia plus Shunts	Elevated	—	—	—	Normal or slightly elevated	Normal	Normal	Low O_2 saturation of arterial *blood*	Angiocardiography ECG chest films	In certain cases it is possible to pass the catheter through the mitral valve and a rudimentary right ventricle *into the LA*
Ebstein Malformation of *Tricuspid* Valve plus Atrial Septal Defect	Elevated during *ventricular* systole	Low or normal	Low or normal	Low or Normal	Normal or *slightly* elevated	Normal	Normal	Low O_2 saturation of arterial blood	Angiocardiography ECG chest films	

TABLE III *Catheterization in Simple Shunts*

Disease	Pressures							Other significant data of catheterization	Other tests which may be useful	Remarks
	RA	RV	PA	PA wedge	LA	LV	AO			
Atrial Septal Defect or Anomalous Venous Return	Normal or slightly elevated (not above 10 mm)	Normal or slightly elevated (usually not above 50 mm)	Normal or slightly elevated (not above 50 mm)	Normal	Normal	Normal	Normal	Higher oxygen content in RA than in the average of SVC + IVC (>1 5 vol %)	ECG Phono chest films (selective angiocardiography during catheterization for pulmonary anomalous venous return)	If RA pressure is markedly elevated there are additional lesions causing RV failure If the RV pressure is above 50 mm, significant pulmonary arteriosclerosis is present
Ventricular Septal Defect	Normal	Normal or slightly elevated (not above 60 mm)	Normal or slightly elevated (not above *60 mm*)	Normal	Normal	Normal	Normal	Higher oxygen content in RV than in RA (>1 0 vol %)	Phono ECG chest films	If RV is markedly elevated there are additional lesions (pulmonic stenosis, overriding aorta pulmonary arteriosclerosis)
Patent Ductus Arteriosus	Normal	Normal or slightly elevated	Normal or slightly elevated	Normal	Normal	Moderate increase of systolic pressure	Moderate decrease of diastolic pressure	Higher oxygen content in PA than in RV and RA (>1 0 vol %)	*Phono* ECG chest films	If RV and PA reveal hypertension there may be a reversed shunt (right-to left) caused by pulmonary arteriosclerosis severe aortic stenosis or coarctation of the aorta

I ulmonic Steno is	Normal	Elevated	Normal or low	Normal	Normal	Normal	Normal	—	Phono ECG chest films Angiocardiogram	There are three types valvular infundibular and combined If it is an infundibular stenosis a typical pattern and a low pressure are found in a separate chamber which is between the main cavity of the RV and the pulmonic valve If it is combined two gradients of pressure are found
I ulmonic In ufficiency	Normal	Normal or slightly elevated	I ow di a tolic pressure	Normal	Normal	Normal	Normal	—	FKY of I A and *Ao I hono* I CG chest films	
Aortic Coarctation (uncomplicated)	Normal	Normal	Normal	Normal	Normal	Elevated	Elevated (above coarctation) decreased (below coarctation)	— —	Retrograde angioaortography I KY of descending Ao I hono ECG I ulse tracings	

[illegible]

Disease	Pressures							Other significant data of catheterization	Other tests which may be useful	Remarks
	RA	RV	PA	PA wedge	LA	LV	AO			
Mitral Stenosis (with minimal or no insufficiency)	*Normal*	Elevated	*Elevated*	*Elevated*	Elevated	Normal (or low)	Normal (or low)	Low cardiac output or lack of increase with exertion	Phono ECG chest films	
Mitral Insufficiency (with minimal or no stenosis)	Normal	Normal or slightly elevated	Normal or slightly elevated	Normal or slightly elevated	Elevated during ventricular systole	Normal or slightly elevated	Normal	—	EKY of LA Phono ECG, chest films	
Aortic Stenosis (with minimal or no insufficiency)	Normal	Normal	Normal	Normal	Normal	High	Normal or low	—	Phono EKY of LV and Ao Pulse tracings (indirect and direct) ECG, chest films	
Aortic Insufficiency (with minimal or no stenosis)	Normal	Normal	Normal	Normal	Normal	Elevated	Elevated *systolic* lowered diastolic	—	Phono, EKY of LV *and* Ao Pulse tracings (indirect and direct) ECG, chest films	
Tricuspid Stenosis (with minimal or no insufficiency)	High	Normal	Normal	Normal	Normal	Normal	Normal	—	Venous pressure jugular tracing	
Tricuspid Insufficiency (with minimal or no stenosis)	High during ventricular systole	Normal or slightly elevated	Normal	Normal	Normal	Normal	Normal	—	Venous pressure, jugular and hepatic tracing ECG, chest films	

Pulmonic Stenosis	Normal	Elevated	Normal or low	Normal	Normal	Normal	Normal	—	Phono ECG chest films Angiocardiogram	The stenotic type valvular infundibular and combined If it is an infundibular stenosis a typical pattern and a low pressure are found in a separate chamber which is between the main cavity of the RV and the pulmonic valve If it is combined two gradients of pressure are found
Pulmonic Insufficiency	Normal	Normal or slightly elevated	Low diastolic pressure	Normal	Normal	Normal	Normal	—	EKG of PA and Ao Phono ECG chest films	
Aortic Coarctation (uncomplicated)	Normal	Normal	Normal	Normal	Normal	Elevated	Elevated (above coarctation) decreased (below coarctation)	— —	Retrograde angioaortography EKG of descending Ao Phono ECG Pulse tracings	

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